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THE UNIVERSITY OF ALBERTA

NEUROPSYCHOLOGICAL FUNCTIONING OF THE ELDERLY WITH DEGENERATIVE BRAIN DISEASE AND DEPRESSIVE DISORDER

BY

© ASHA SINHA

A THESIS

SUBMITTED TO THE FACULTY OF GRADUATE STUDIES AND RESEARCH
IN PARTIAL FULFILMENT OF THE REQUIREMENTS FOR THE DEGREE
OF DOCTOR OF PHILOSOPHY

DEPARTMENT OF EDUCATIONAL PSYCHOLOGY

EDMONTON, ALBERTA

FALL, 1987

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ISBN 0-315-41039-6

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DEGREE: DOCTOR OF PHILOSOPHY

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Asha Sinha
.....
(Student's signature)

.....
(Student's permanent address)
14848-64 AVE.....
EDMONTON, ALBERTA
T6H-4Y4

Date: June 17, 1987

THE UNIVERSITY OF ALBERTA

FACULTY OF GRADUATE STUDIES AND RESEARCH

The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies and Research for acceptance, a thesis entitled NEUROPSYCHOLOGICAL FUNCTIONING OF THE ELDERLY WITH DEGENERATIVE BRAIN DISEASE AND DEPRESSIVE DISORDER submitted by Asha Sinha in partial fulfilment of the requrirements for the degree of Doctor of Philosophy in Educational Psychology.

W Hornmidt
Supervisor

Kathleen A. Allen

D McLeod

Robert Franke

Edward Jacobs

Date April 28, 1987

DEDICATION

I would like to dedicate this dissertation to my husband, Dr. Birendra K. Sinha, who has been extremely supportive, understanding and encouraging throughout my undergraduate and graduate studies.

Also, I would like to dedicate this dissertation to the memory of our beloved dog, "Zippy", who taught me determination, patience and forgiveness.

ABSTRACT

Cognitive impairment in the elderly suffering from degenerative brain disease (Alzheimer's Disease and Multi-infarct Dementia), and Depression is the focus of this study. The rationale of this research was based upon the difficulty in differential diagnosis of dementia and depression due to their overlapping symptoms. A comprehensive neuropsychological test, The Luria-Nebraska Neuropsychological Battery (LNNB) (Golden et al., 1980) was used to examine the neuropsychological functioning of the above-mentioned patient groups. Four other tests commonly employed for assessing brain impairment were also used: Halstead-Wepman Aphasia Screening Test, Trail Making Test, Wechsler Memory Scale, and Wisconsin Card Sorting Battery. The normal comparison consisted of elderly persons from two age groups: (a) young elderly (55 to 74), and (b) old elderly (75 and over).

It was hypothesized that the elderly suffering from Alzheimer's Disease (AD), Multi-infarct Dementia (MID) and Depression (DEP) would show significant neuropsychological impairment on the tests. Specifically, it was expected that the AD patients would show global and uniform impairment on all neuropsychological measures, whereas the MID patients would show focal and localized impairment. Compared to the AD and MID patients, the depressed elderly were expected to perform better on all tests. No significant difference between the normal young elderly and normal old elderly was expected.

The results show that the AD and MID groups performed significantly worse ($p < .0005$) on all neuropsychological measures compared to the normal controls and the depressives. As expected,

none of the obtained scores of the DEP group fell in the organic range, although compared to the normal control groups, the depressed patients performed poorly on most neuropsychological measures. The expectation that the AD and the MID groups would perform significantly differently was not confirmed. In fact, both the groups revealed similar patterns of test performance. As expected, the performance of the old elderly and the young elderly did not fall in the impaired range. There were few differences in the normal controls on the basis of age. Thus, the neuropsychological tests did differentiate the dementia groups from the depressive group, but failed to differentiate the two dementia groups (AD and MID).

ACKNOWLEDGEMENTS

I wish to express my sincere thanks to my supervisor, Dr. W.H.O. Schmidt for his interest, support and guidance in all stages of my dissertation research. Dr. Schmidt is not only a scholar, he is also a very understanding and encouraging advisor.

I am very thankful to other members of my committee, Dr. D. Skelton, Dr. R. Frender, Dr. T. Nelson, and Professor K.A. Dier, for their helpful comments during the preparation of the dissertation. I thank Mr. C. Humphrey of the Department of Computing Services for his help and suggestions in data analyses.

I am particularly indebted to Dr. E.E. Fox, Associate Chairman, Department of Educational Psychology, who took personal interest in providing administrative support in completing this dissertation.

I would also like to thank the clinical and administrative staff of the Edmonton General Hospital, Misericordia Hospital and Alberta Hospital of Edmonton for providing appropriate subjects, space and other necessary facilities for collecting the data. Help of the Alzheimer's Society and the Society for Retired and Semi-Retired of Edmonton is greatly appreciated. I am also thankful to Anita Moore for her help in typing the manuscript.

I would like to express my gratitude to all those elderly persons who volunteered to participate in this research. The cooperation of their family members deserves special recognition.

Finally, I am indebted to my husband, Dr. Birendra K. Sinha, and my sons, Animesh, Neel and Smit, for their unconditional support

and understanding. Our family dogs "Zippy" and "Sona" were special sources of joy during many frustrating hours of work.

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CHAPTER I

INTRODUCTION

The 1981 Census of Canada reports that 9.7% of the Canadian population are 65 years of age and older (Statistics Canada, 1984). It is estimated that by the year 2001, persons who are 65 years and over will constitute nearly 12% of the total population of Canada. In the United States the older population is expected to increase about 16% by the year 2010 (United States Bureau of the Census, 1975). A similar trend of increasing population of older persons has been noted in European countries (Marquis Academic Media, 1977).

The geriatric literature is replete with studies implicating age as one of the major factors in a wide variety of medical, neurological, behavioral and emotional problems (Butler & Lewis, 1982; Botwinick, 1984; Poon, 1980; Zarit, 1980). Specifically, there is growing concern for the possibility of extensive as well as intensive neuropsychological deficits among the aged (Fuld, 1983; Gainotti, Caliagirone, Masullo, & Micell, 1980; Karl, 1982). High risk of neuropsychological problems in the elderly has generally been acknowledged by behavioral scientists, physicians, and mental health professionals (Birren & Schaie, 1977; Botwinick, 1984; Butler, 1982; Tresch, Folstein, & Robins, 1985; Zarit, 1980). Because the elderly population (65 years and over) is growing at a faster rate than any other age group, there is a need for comprehensive and accurate assessment of neuropsychological functioning of the aged in order to develop appropriate treatment and management plans (Birren & Renner,

1980; Johansen, Gustafson, & Reisberg, 1985; Reisberg, 1983; Rosen & Mohs, 1982).

Although there is a high risk of neuropsychological problems among the aged, early detection may prevent or slow down the impending cognitive and behavioral dysfunction in mild cases. Even in medical conditions that produce serious neuropsychological impairment requiring continuing hospital care, accurate evaluation of cognitive deficits is important in management and rehabilitation. With their expertise on brain-behavior relationships, neuropsychologists are expected not only to judge the presence or absence of cerebral impairment, but also to assess the severity, localization, etiology, and behavioral implications of various degenerative brain disorders. However, the assessment of neuropsychological functioning of old persons is extremely difficult because of the complex interaction of disease and advancing age. Consequently, intensive research on the assessment of neuropsychological changes in geriatric patients is essential for diagnostic efficiency, treatment, and rehabilitation.

Rationale and Significance

Many studies suggest that there is considerable increase in neuropsychological and psychiatric problems with advancing age. However, there is no consistent finding about the nature and degree of cognitive and affective change associated with age (Albert, 1981; Bak & Green, 1980; Benton, Eslinger, & Damaso, 1981; Bigler, Steinman, & Newton, 1981). Detection and evaluation of pathological changes in the aged are complicated primarily by difficulties in research design, instrumentation, norms, and medical diagnosis. Availability,

motivation, and health status of the subjects along with ethical considerations introduce additional complexity into the collection, analysis, and interpretation of the data (Butler, 1980; Lawton, Whelihan, & Belsky, 1980; Raskin & Jarvik, 1979; Zarit, 1980).

Since varying degrees of neuropsychological impairment have been observed in old persons (Albert, 1981; Brinkman, Largen, & Gerganoff, 1983; Fabry, 1982; Masur, 1980; Price, Fein, & Feinberg, 1980; Schuldermann, Schuldermann, Merryman, & Brown, 1983; Schwarts, 1982; Sulkava, 1982), it would be beneficial, both theoretically and clinically, to compare the neuropsychological performance of normal healthy (physically and mentally) elderly persons with the performance of those suffering from specific neurological and psychiatric diseases. In the present research, two commonly diagnosed medical conditions in the elderly population, degenerative brain disease (i.e., Alzheimer's disease and Multi-infarct dementia), and Depression have been studied to determine the amount and nature of neuropsychological changes in persons with these medical conditions. In addition, the neuropsychological functioning of two elderly groups, the young elderly (55-74) and the old elderly (75+) are examined for comparison purposes.

The rationale for selecting Alzheimer's Disease (AD), Multi-infarct Dementia (MID), and Depression (DEP) in the present research is related to the fact that these conditions are most commonly observed by physicians and psychiatrists dealing with elderly patients (Magni, Ed Leo, & Schifano, 1985; Tresch et al., 1985). A great deal of confusion exists in the literature regarding the precise nature of neuropsychological deficits in these conditions. As will be

discussed in the Review of Literature section, deterioration of cognitive functions, such as attention, memory and psychomotor retardation have been observed in degenerative brain disease as well as in depressive disorder (Copeland, Kelleher, Kellett, Fountain-Gourlay, Cowan, Barron, & DeGruchy 1974; Harris, 1985; Lauter, 1985; Von Ammon Cavanaugh, 1983). Moreover, similar cognitive deficits are manifested in treatable dementias and pseudodementia conditions (Blazer & Williams, 1980; Gustafson, 1985).

The differential diagnosis of Alzheimer's Disease (AD), Multi-infarct Dementia (MID) and Depression (DEP) is generally made by physicians primarily on the basis of clinical features supplemented by some neuropathological and neurochemical tests (Perry & Perry, 1985). However, the research literature clearly demonstrates the fact that precise differentiation of cognitive deficits in various types of dementia and geriatric depression is extremely difficult (Eisdorfer, Cohen, & Keckich, 1983; Gibson, 1981; Katzman, 1982; Kuhl, Metter, Riege, & Hawkins, 1985; Ladurner, Pieringer, & Sager, 1981; Straker, 1984). For example, Garcia, Reding and Blass (1981) report that a 10% to 50% error rate is observed in distinguishing early dementia from depression or early aging changes. Since there is no specific biochemical or radiological marker for degenerative brain disease, a complete neuropsychological assessment may be helpful in differential diagnosis (Kuhl et al., 1985).

An important issue in geriatric neuropsychology is the selection of measuring instruments with adequate reliability and validity for making accurate comparisons between various elderly groups (Birren & Sloane, 1980; Schwarts, 1982; Welden, 1983).

Although there are many neuropsychological tests available, most of them are limited in scope and have an inadequate psychometric data base (standardization, norms, reliability and validity). None of the available neuropsychological assessment devices has been standardized and normed for the elderly population. Recently neuropsychologists have shown great interest in the Luria-Nebraska Neuropsychological Battery (LNNB) which is currently the most comprehensive test battery available (Golden, Hammeke, & Purisch, 1980). The Luria-Nebraska is a theory-based test instrument that assesses a variety of cognitive abilities. In a recent article, Erlandson, Osmon, and Golden (1981) asserted:

> The Luria Battery, then provides a more exhaustive evaluation of cognitive processes, one which corresponds to the way in which the brain theoretically carries out the cognitive process. The more precise delineation of brain dysfunction provided by the Luria-Nebraska may allow for more powerful statements to be made concerning the relationship of brain function to personality (p.146).

Golden et al. (1981) have suggested that the Luria-Nebraska is an appropriate instrument for the assessment of neuropsychological functioning in the elderly. In a general review paper, Gillen, Golden, and Eyde (1983) present empirical as well as theoretical reasons for the use of the Luria-Nebraska with elderly populations. MacInnes, Golden, Grable, Cole, Uhl, and Greenhouse (1982) have demonstrated that the normal elderly who are living independently perform as well as normal adults on the LNNB. Therefore, they suggested that brain damage in the elderly population is likely to be detected with greater accuracy through the LNNB than through any other neuropsychological test presently available.

Since the Luria-Nebraska has been in use for only a few years, there is a great need for psychometric research on this instrument, particularly for the purpose of establishing its usefulness with the elderly population suffering from various neurological and psychiatric disorders. It should be noted that Golden and his associates (1978, 1980, 1981), have published empirical evidence to support the LNNB's efficacy in detecting specific neuropsychological dysfunctions in humans. However, a recent comprehensive review of this test by Stambrooks (1983), based on his doctoral dissertation, concluded that considerably more research is required on clearly defined independent populations of elderly persons. Similar observations regarding the need for additional data from different pathological groups have been made by other researchers (Adams, 1980; Crosson & Warren, 1982; Sears & Hirt, 1984; Snow & Hynd, 1984).

The present research also aimed to examine the performance of the subjects on four other neuropsychological tests commonly used for the assessment of cognitive impairment. They are: (a) Halstead-Wepman Aphasia Screening Test (1959), (b) Trail Making Test (Reitan, 1958), (c) Wechsler Memory Scale (1945), and (d) Wisconsin Card-Sorting Test (Berg, 1948). These additional tests assess the following neuropsychological functions measured by the LNNB: motor functions, visual functions, receptive speech, expressive speech, writing, reading, arithmetic, memory and intellectual processes. Selection of these functions was guided by a recent recommendation by Albert (1981) regarding useful assessment areas in geriatric neuropsychology. He asserted that adequate neuropsychological assessment of the elderly must include at least five functions: attention, language, memory,

visuo-spatial ability, cognitive flexibility and abstraction. These functions are also measured by the four other widely-used neuropsychological tests included in this study. Table 1 shows the neuropsychological functions measured by the Luria-Nebraska Neuropsychological battery and other tests selected in this research. The rationale for employing four tests in addition to the main comprehensive battery (the Luria-Nebraska) was to check whether the deficits in selected neuropsychological functions would be confirmed by similar measures.

The significance of the present research is apparent when we consider the fact that certain forms of systematic medical disease, such as diabetes, lung disease and hypertension also adversely affect cognitive functioning in a manner similar to that of the brain disorders. Similarly, toxic-metabolic disturbance and some psychiatric disorders, most commonly found in the elderly, such as depression also produce symptoms that can be mistaken for nonreversible cognitive changes (Fuld, 1984; Gainotti, Caliagirone, Masullo, & Micell, 1980; Koszniak, Carron, & Fox, 1979). The nature of cognitive deficiency in dementia and other medical conditions is difficult to understand without adequate assessment methods. Further, it is hard to distinguish between the cognitive decline found in dementia and depression without the help of a refined measuring instrument. Accurate diagnosis is critical for instituting successful treatment programmes for either dementia or depression. This research should also provide a better understanding of the nature, level and degree of organic impairment in specified groups of elderly persons.

The present research is significant also because to date there

Table 1

Neuropsychological Functions Measured by the

Luria-Nebraska and Other Tests

Selected Scales on the Luria-Nebraska Neuropsychological Battery	Other Tests
1. Motor functions	Halstead-Wepman Aphasia Screening Test Trail Making Test
2. Visual functions	Trail Making Test Wisconsin Card Sorting Test
3. Receptive Speech 4. Expressive Speech 5. Writing 6. Reading 7. Arithmetic	Halstead-Wepman Aphasia Screening Test
8. Memory	Wechsler Memory Scale
9. Intellectual Processes	Wisconsin Card Sorting Test

have been only a few published studies based on systematic testing concerning neuropsychological changes in the elderly (Poon, 1980; Raskin & Jarvik, 1979; Zarit, 1980). Moreover, these few studies have employed only a very small number of tests, thereby rendering the findings limited in scope (Golden, 1981). In the present study a variety of well-established instruments were used.

Another unique feature of the present research is that the Luria-Nebraska Neuropsychological Battery (LNNB) has been used for the first time on well-defined groups of the aged with specific degenerative brain disease and psychiatric disorder. Although there are some studies on the LNNB using elderly subjects (Gillen, et al., 1982; MacInnes, et al., 1982; Spitzform, 1982), these are mainly concerned with developing test norms of the elderly, rather than examining the nature of cognitive deficit or the differential pattern of neuropsychological functions. Since the LNNB is a standardized, comprehensive and reasonably valid instrument requiring relatively short time to assess numerous specific cognitive abilities (Golden, 1981), the findings of the present research have many clinical applications with respect to the geriatric population. The results of this research should be helpful to geriatric neuropsychologists in interpreting test scores; they are also likely to assist physicians in diagnosing neurological disorders with overlapping symptoms as well as in providing adequate treatment, management, and rehabilitation facilities.

In summary, the present research used a well-standardized, comprehensive neuropsychological test, namely the Luria-Nebraska Neuropsychological Battery (LNNB) to measure the precise nature and

amount of neuropsycholgical deficits in older persons suffering from degenerative brain disease and depressive disorder. Only Alzheimer's Disease (AD) and Multi-infarct Dementia (MID) were selected to represent the degenerative brain disease. As for the depressive disorder, only Major Depression as defined in DSM III was included in the study. These disorders were selected because of their high prevalence and overlapping symptomatology. Thus, this research provided comparative neuropsychological data on selected groups of elderly patients as well as on two age groups--young elderly (55-74 years) and old elderly (75 years and above) of normal controls.

CHAPTER II

REVIEW OF THE LITERATURE

Normal Aging Processes

Definition and Types of Aging

In order to evaluate an abnormal aging process, it is important to understand the process of normal aging. Perhaps the most striking observation is that progressive and irreversible changes occur throughout the life span. However, these age-associated changes do not seem to follow any typical pattern.

From a biological point of view, the aging process has been defined as "...an internal biological determinant that gradually alters appearance, physical capacity and behavior" (Zarit, 1980, p. 7). However, this biological explanation of aging has been criticized as being simple, narrow and misleading. A variety of social, psychological and environmental factors are also involved in the process of aging. Thus, aging is a complex phenomenon which cannot be explained in isolation. According to Birren (1959), the aging process should be conceptualized at three levels: the level of (1) biological aging, (2) psychological aging, and (3) sociological aging.

Biological aging refers to changes that occur in the structure and functions of the body organs (tissues and cells) over time. External appearance reflects the most obvious and noticeable physiological change as people grow older. Psychological aging refers

to changes in cognition, affect, personality, and behavior over time which occur due to both life experience and biological changes. It should be noted that, like biological aging, psychological aging also shows complex and individualized patterns of change. Sociological aging is concerned with changes in norms, expectations and roles of a person in the family and in the society at large (Atchley, 1980; Bornstein & Smircina, 1982).

These three different aging processes do not occur in isolation. Normal aging is based upon the interaction of all the three processes. It is an implicit assumption that the process of aging involves a long and gradual decline in all the three areas (Neugarten, 1975; Zarit, 1980). However, the age at which declines are manifested and the extent of change may vary considerably from person to person (Botwinick, 1984; Butler & Lewis, 1982). Individual differences in aging have been attributed to genetic endowment, psychological factors, and social conditions. Among the psychological factors, personal motivation, opportunity for learning, and attitude toward life seem to have major influence on the aging process (Butler & Lewis, 1982; Schaie, 1983).

Popular myths and stereotypes about aging are generally negative and uncomplimentary. Perhaps the most damaging myth is the myth of "senility" based on the assumption that mental abilities must show progressive deterioration as a result of a "natural" biological aging process. Specifically, forgetfulness, motor and perceptual difficulties, general intellectual decline, inability to learn, lack of motivation, anxiety, depression, short attention-span, and confused thinking are all considered major characteristics of normal aging

(Atchely, 1980; Brubaker & Power, 1976; Hess, 1974; Quadagno, 1980). However, as we learn more about aging, we may be able to explain why some aged persons show little or no deterioration in their cognitive and social abilities well into their 80s and 90s (Thompson & Marsh, 1973).

Structural and Neuronal Changes

It has been noted that with advancing age, there is a gradual change in the size and weight of the brain due to neuronal loss. Loss of neurons is considered the normal effect of aging. It is estimated that by the age of 90 there is a loss of approximately 45% to 50% of cells in the cerebral cortex (Bondareff, 1977; Brody & Vijayashanker, 1977). Since these cells do not undergo mitosis, the resultant loss of neurons is likely to affect not only the higher mental functioning that depends on the central nervous system, but also personality functions of the individual. Cortical thinning as a result of diminished extracellular space is also a factor that affects the cognitive functioning of the individual (Bondareff, 1981).

Other identified changes in the brain associated with normal aging are a) deepening and widening of the fissures of the brain, b) degeneration of Schwann cells, c) decrease in cerebral blood flow, and d) flattening of the brain surface. In addition, the amount of lipofuscin within the cells increases and neurofibrillary tangles and neuritic plaques start to expand. However, it should be noted that neuronal loss, neurofibrillary tangles, neuritic plaques and lipofuscin are present in elderly persons undergoing the normal aging process as well as in those with degenerative brain disease. The

difference between the two is that in normal aging these changes are observed only in small amounts.

Sensory Changes

Changes in various sensory functions, such as vision and hearing commonly occur with advancing age. The progressive bilateral loss of hearing of high frequency tone known as presbycuis occurs frequently with advancing age. Approximately 13% of aged persons have high frequency tone impairment and it is more prominent in men (Corso, 1977). It is also most progressive after age fifty-five and has implications for speech comprehension (Kryter, 1960; Spoor, 1967). In their study of hearing ability and intellectual test performance, Granick, Kleben, and Weiss (1976) found that the hearing ability affects psychological test performance. They concluded that the elderly are likely to be more capable cognitively than their test performance suggests. With respect to vision, changes in the eyes due to aging are likely to produce decreased visual acuity, impaired near-distance vision, loss of sensitivity to colors, increased sensitivity to glare and reduced peripheral visual fields.

Research suggests that sensory deficits may affect language comprehension and interpersonal communication abilities (Fozard & Popkin, 1978). The loss of hearing and vision has considerable effect on cognitive functioning of the elderly (Botwinick & Storandt, 1974). Since almost all procedures for assessing mental ability require adequate vision and hearing, sensory deficits are likely to lower the I.Q. scores of the elderly. In other words, poor performance in

cognitive tasks may be due to sensory loss in advancing years, rather than cognitive decline.

Changes in Cognitive Functioning

Over the past 20 years gerontological researchers have paid considerable attention to the problem of age differences in cognitive functioning (Baltes & Schaie, 1974; Horn & Donaldson, 1977; Schaie & Baltes, 1977; Willis & Baltes, 1980). It is generally assumed that cognitive processes undergo gradual change with advancing age. Cognitive decline has been suggested in intelligence, memory, perception, learning and problem-solving (Bak & Green, 1980; Benton, Eslinger, & Damaso, 1981). However, there is a great deal of disagreement among researchers about the pattern, nature and amount of deficit that occurs in various cognitive processes on account of age. A brief review of the literature will focus upon research findings pertaining to the cognitive processes of the normal elderly.

Intelligence

In the past it has been widely believed that intellectual decline in old age is inevitable. However, the evidence is contradictory. According to Wechsler (1958) intelligence, as measured by intelligence tests, reaches its peak between ages 18 and 25, and thereafter declines; both Wechsler (1971) and Cattell (1971) provided evidence that this decline is gradual at first but accelerates with advancing age. Botwinick (1977, p. 580) also states that "decline in intellectual ability is clearly part of aging picture", but admitted that these changes may start very late in life, and may be small and involve only few areas of mental functioning. Baltes and Schaie

(1974) have come to believe that "general intellectual decline in old age is largely a myth".

Another controversial issue is whether or not people with initially higher intelligence show less decline with age. Some studies reported that bright people show less decline with age (e.g., Riegel, Riegel, & Meyer, 1967) while others have suggested a similar rate of decline (Eichorn, 1973; Eisdorfer & Wilkie, 1973). Blum and Jarvik (1974) observed that those who were initially more able and better educated showed less decline. Williamson, Munley, & Evans (1980) conclude that people who use their knowledge or keep their mind active through reading, writing and exchange of information are less likely to suffer intellectual decline.

Evaluation of Intelligence. Differences in findings regarding decline of mental functioning can be attributed, in part at least, to differences in procedures for assessing intelligence. Although adult intelligence has been defined in many ways, there are two main approaches to the study of intellectual behavior (Botwinick, 1977; Sternberg & Detterman, 1979). The first, more dominant approach, employs a psychometric concept of intelligence. This approach has developed with intelligence testing emphasizing the traditional concept of human intelligence and prediction of behavior (Cattell, 1971; Horn & Donaldson, 1976). Another approach considers intelligence in terms of perception, memory, and problem-solving (Sternberg & Detterman, 1979).

Gerontological researchers have mainly used psychometric methods to measure the intellectual behavior of the elderly (Cattell, 1971; Horn, 1977; Wechsler, 1958). Since World War II, the Wechsler

Adult Intelligence Scale (WAIS), which measures both verbal and nonverbal skills, has been widely used in aging research (Baltes & Labouvie, 1973; Doppelt & Wallace, 1955; Eisdorfer & Cohen, 1961). Relevant research on this topic reveals that verbal intelligence remains intact for a long time, but performance intelligence starts to decline more rapidly with advancing age (Eisdorfer & Cohen, 1961; Overall & Gorham, 1972). In fact, some verbal skills (e.g., vocabulary) have been reported to improve with age (Williamson, Muney, & Evans, 1980). Gradual intellectual decline in some areas measured by the verbal subtests has been noted only after the age of 70 (Krammer & Jarvik, 1979). Relatively greater decline has been observed on the Performance scales of WAIS, especially on those subtests that require visual-motor-spatial and visual-motor-perceptual abilities (e.g., digit symbol, block design and object assembly). Discrepancy between verbal and performance scores has been found so consistently that it is labelled "the classic aging" pattern (Doppelt & Wallace, 1955; Wechsler, 1958).

Crystallized and Fluid Intelligence. Cattell (1971) has conceptualized intelligence in terms of (a) crystallized and (b) fluid dimensions. With reference to the WAIS, crystallized intelligence is similar to what is assessed in the Verbal tests, whereas fluid intelligence is similar to what is assessed in the Performance tests. Cattell describes crystallized intelligence as a product of intentional learning within the context of the individual's cultural and educational background. On the other hand, fluid intelligence refers to perception of relationships, thinking, abstract learning, concept formation, and problem solving. It is suggested that

crystallized and fluid intelligence both increase simultaneously up to the age of 15. However, fluid intelligence starts to decline after 15 through age 60, after this age it stabilizes. Decline in the fluid intelligence has been attributed to changes in various aspects of physiological and neurological functioning that occur with the aging process. Crystallized intelligence, on the other hand, stabilizes by mid-adolescence. There is also a slight increase in crystallized intelligence with age (Williamson, Munley, & Evans, 1980). Not only do different methods of assessing intelligence lead to differing pictures of the nature of intellectual changes that come with increasing age, but research designs also influence the findings. In this connection the difference between findings based on cross-sectional and longitudinal research need to be highlighted.

Cross-sectional vs. Longitudinal Research. Both longitudinal and cross-sectional methods have been used to shed light on the nature and pattern of cognitive decline with age. These two methods have provided contradictory patterns of change in intelligence with advancing age. Cross-sectional studies have reported consistently lower scores among older subjects on both Verbal and Performance measures. Poor performance by the older subjects on the WAIS Verbal scale has been noted on the information, comprehension, and vocabulary subtests, all of which rely primarily on prior learning. They also scored low on the Performance subtests of the WAIS and Raven's Progressive Matrices test (Botwinick, 1977). However, longitudinal studies have demonstrated greater stability in intelligence scores than expected. Only very little decline in verbal abilities was noted before age 70. Even after age 70, the decline was not that severe.

Somewhat greater decline with age was observed on tasks involving novel responses (performance subtests of WAIS), but the decline was less than that reported in cross-sectional research (Blum, Clark, & Jarvik, 1973; Wilkie & Eisdorfer, 1973).

The discrepancies in the findings may be explained as due to methodological differences in the cross-sectional and longitudinal designs. Since different age agroups are studied at one time in the cross-sectional approach, it is difficult to distinguish the precise contribution of aging from the influence of education, socioeconomic background, ethnicity, etc. By contrast, the longitudinal method provides a more precise evaluation of age-related changes in cognitive functioning, because subjects in the same age group are studied over time.

It should be noted that performance on cognitive tasks reflects the interaction of a variety of organismic and environmental variables (Baltes & Labouvie, 1973). For example, different educational and occupational background of the elderly must be taken into account in assessing intellectual abilities. It has been observed that socioeconomic status, as indicated by education and occupation also affect performance of older adults on intelligence tests (Gribbon, Schaie, & Parham, 1980). The elderly who come from lower socioeconomic background and have health problems usually show greater age related decline in their performance on psychological tests (Schmitz-Scherzer & Thomae, 1983). Cohen (1979) found that in comparison with young adults, age differences on intellectual test performance were much smaller for the elderly with college education than for those with grade school education. Furthermore, incidental

learning and problem-solving abilities are also affected by educational background (Arbuckle, Gold, & Andres, 1986; Schaie, 1983).

Memory

The relationship between memory and age has become a major issue in the field of gerontology over the past decade (Kausler, Lichty, & Davis, 1985). It is evident that research relating to the memory processes of the elderly has not only practical significance, but also implications for theoretical conceptualization and direction for future research (Kausler & Hakami, 1983; Fozard, 1980). Although it is generally assumed that with advancing age decline in memory occurs rapidly, the extent of memory loss in the healthy elderly has probably been exaggerated by both the elderly themselves and by those who work with them, including professionals (Zarit, 1980).

Several studies suggested that problems with memory are common complaints among the elderly. Lowenthal & Berkman (1967), in a community survey, found that about one-half of persons over 60 complained about memory. In another survey, Kahn et al. (1975) reported that two-thirds of a sample of older psychiatric patients had similar complaints. However, performance on objective memory tests did not correlate with the reported complaints (Gurland, Dean, Crow, & Golden, 1980; Kahn et al., 1975).

In the following section various aspects of memory, such as storage and retrieval, sensory memory, primary memory, and secondary memory will be reviewed briefly.

Storage and Retrieval. Studies on memory are mostly concerned with storage and retrieval processes. Storage refers to placement of information in the memory, whereas retrieval process is concerned with

bringing back the stored information into awareness. Most findings indicate that the storage mechanism remains relatively intact with age, but decline occurs in the retrieval mechanism (Burke & Light, 1981; Hultsch & Pentz, 1981; Park, Puglisi, & Sovacool, 1984; Tulving, 1983). Several explanations have been offered for this decline, such as poor utilization of contextual information, and poor organization of semantic memory and information overload (Criak, 1977, 1982, 1984).

Sensory Memory. Recently sensory memory has received the attention of researchers. Essentially, it is perceptual in nature and is concerned with registration of external stimuli (e.g., visual and auditory) in memory for very short periods. Sensory memory lasts only for milliseconds. Several studies on sensory memory of the elderly have shown considerable impairment with age. Walsh & Thompson (1978) found that the age difference was greatest when the stimuli were presented for only short periods of time (e.g., 50 milliseconds). Out of 10 aged persons, only two showed sensory memory, whereas 9 out of 9 young persons were able to identify the stimuli. Similar findings have been reported by Kline & Szafran (1976). However, these researchers as well as Salthouse (1976) noted that when presentation time was increased to 100 milliseconds, very little difference in sensory recall occurred between old adults and young persons. Similar results were obtained by Abel (1972) who reported that when duration of exposure to the stimuli was increased to 500 milliseconds, there was very little difference in sensory recall between young and old persons. It has also been observed that memory for meaningful items remain intact for a long time (Ferris, Crook, Clark, McCarthy, & Raie, 1980; Park et al., 1986).

Primary Memory (Short-term). Primary memory refers to the temporary storage capacity for information that is consciously and actively being processed at a given time. Measurement of primary memory is made through free recall tasks and tasks involving immediate memory span. Studies in this area suggest that the holding process in primary memory is not affected by advancing age (Craik, 1968, 1977, 1984). Review of the literature suggests that primary memory remains stable over time as long as no recognition of the stimuli is required (Craik, 1977; Drachman & Leavitt, 1972). For example, performance on the digit span forward task does not decline with age but performance was somewhat poor on the digit span backward task where recognition of the stimuli is required (Botwinick & Storandt, 1974; Walsh, 1975).

Secondary Memory (Long-term). Secondary memory is concerned with transfer of material from primary memory into a storage system that can be retrieved and used over and over again. Research findings suggest an age difference in acquisition of new information and retrieval of this stored information (Craik, 1977; Craik & Robinowitz, 1984). Acquisition and retrieval processes of memory have been measured by recall and recognition tasks in both old and young persons. The elderly have been found to perform consistently poorly on all recall tasks, but performance on recognition tasks is controversial. Some researchers report similar performance on recognition tasks by both the old and young (Park, Puglisi, & Sovacool, 1984; Rabinowitz, Craik, & Ackerman, 1982; Schonfield, 1965), while others report decrements in both recall and recognition tasks in the elderly compared to the younger age group (Botwinick & Storandt, 1974; Perlmutter, 1978). Craik (1977) and Smith (1980) have

reviewed the studies on retrieval difficulty in the memory of older and younger subjects, and concluded that retrieval difficulty is not the only cause of age difference in recall. Difficulty in encoding the information has also been suggested as the cause of memory deficit (Craik & Simon, 1980; Smith, 1980).

Factors Affecting Memory Functioning. Empirical studies have attempted to identify factors affecting the memory of the elderly persons. Different explanations have also been presented. Most researchers assume that age difference in memory result from differences in the way older and younger people process information, which is called "processing deficiency". It is believed that older people utilize poor information processing strategies in organizing new incoming information in the primary memory (Craik & Simon, 1980; Smith, 1980). Several techniques have been applied to improve the processing strategy of older subjects. It has been concluded that "effective" processing strategies have not been successful in bringing older person's memory to the level of younger persons (Poon, Fozard, & Walsh-Sweeney, 1980).

Several studies also suggest major differences in memory at the point of acquisition between young and old persons. Researchers believe that it is not that they lose more information, instead they do not know how to properly acquire the information (Botwinick, 1978; Craik, 1977). Although well-documented empirical evidence is not available at the present time, it has been suggested that age-related slowing of mental processing is the major factor in different memory functioning of old and young persons (Salthouse, 1980; Waugh & Barr, 1980). Physiologically oriented explanations of memory deficit have

been presented by Waugh and Barr (1980), and by Salthouse (1980). They disagree with the notion that processing strategies are under the control of individuals. They claim that it is the result of general slowing in the speed of the behavior due to changes in the nervous system (Waugh & Barr, 1980). Several studies have shown that older people require 30% more processing time compared to younger persons, especially with visual sensory information (Walsh & Thompson, 1978;). But in a recent study by Walsh and Prasse (1980), older adults compared to younger subjects required as much as 80% longer processing time from primary to secondary memory. However, the notion of deficiency in processing strategies and slowness in the speed of information processing are still controversial issues among researchers. For example, changes have not been reported in the speed of retrieval for semantic memory and for naming pictures (Craik & Rabinowitz, 1984; Eysenck & Eysenck, 1979; Schaie, 1980).

The role of attention in old age memory has also received some attention in recent research. Several researchers point out that age-related decrement in attention is the cause of memory deficit among the elderly. The effect of attentional deficit has been observed in the performance of the elderly on a number of tasks (Craik & Simon, 1980; Craik, 1982; Hoyer & Plude, 1982; Plude & Hoyer, 1985). However, research on attention and aging is still very limited.

Learning

Learning is another aspect of the cognitive process. It is concerned with acquisition of novel responses or messages. Distinction between memory and learning is difficult to make. In general, learning involves getting information in and memory is

concerned with keeping the information retained for future use (Botwinick, 1981). It has been acknowledged that the learning process is affected by both intelligence and memory (Karl, 1978).

Studies have shown that, in general, old persons do poorly in learning of new materials (Botwinick, 1981). However, it has been suggested that the learning process in the elderly is influenced greatly by such factors as stimulus pacing, interference, and time (Kausler, 1983). It has been found that older people did less well when stimuli were presented with fast speed and had limited time to respond (Plude & Hoyer, 1981). Arenberg (1965) found that when paired associate tasks were presented at a fast rate, the elderly were disproportionately poor in learning the items, but when they were presented at a slow rate, no difference was found among young and old subjects. Similar results have been reported in other studies (Arenberg & Robertson-Tchabo, 1977; Kinsbourne & Berryhill, 1972). However, some investigators have suggested that it is important not only to present stimuli at slow rates, but also to give ample time to the elderly for making responses (Monge & Hultsch, 1971).

Interference has been identified as one of the variables producing learning deficits in the elderly (Arenberg, 1977). But Craik (1977), on the basis of his review of literature, suggests that effects of interference in learning are the same for both young and old persons. It should be noted that age-related changes in learning abilities are generally very small.

Problem-solving

It has been generally assumed that older persons are less capable of solving complex problems as a result of intellectual

decline, reduced memory, slow information processing and inflexibility of thought and planning (Botwinick, 1977; Rabbit, 1977). Most studies suggest that difficulties experienced by the elderly in solving problems are due to lack of flexibility in their problem-solving strategies (e.g., Friend & Zubek, 1958; Wetherick, 1965). However, a few studies do not attribute problem-solving difficulties of the elderly to their inflexible approach and difficulty in shifting attention (Arenberg, 1982; Rogers, Keys, & Fuller, 1976). These studies suggest that older people do not show difficulty in learning if the tasks are meaningful (Park, Publisi, & Smith, 1986).

Redundancy in information seeking behavior has been observed to affect problem-solving skills. Older persons have a tendency to make more irrelevant than relevant inquiries (Denny & Denny, 1974; Denny, 1982). Some researchers assert that the elderly, in general, have problems in solving abstract tasks, but have no difficulty when tasks are concrete (Botwinick, 1978; Rabbit, 1977). However, Arenberg (1974) does not support this conclusion.

It is also believed that the elderly do not use new information in solving problems. They approach the problems through previous knowledge (Kart, Metress, & Matress, 1978). But Watson (1982) disagrees with this interpretation on the ground that it is a general tendency throughout the course of life to select and apply previously acquired knowledge in solving problems. Hence, it is not unique to the elderly.

Within-Group Norms for the Elderly

Caution should be exercised in arriving at a definte conclusion

about the nature of cognitive functioning in the aged on the basis of psychological and neuropsychological tests. The primary reason for this caution is the lack of adequate age-related norms for psychological tests for the elderly. The available normative data for psychological and neuropsychological tests are primarily based on the performance of young adults. Also, at present there is not a single psychological test that is based on systematic comparisons of the normal versus pathological performance of the elderly. Thus, in the absence of adequate normative data for the elderly population, most older subjects are likely to be incorrectly classified as brain-damaged on various neuropsychological test batteries (Zarit, 1980).

Lack of adequate age norms for the elderly in psychological and neuropsychological tests is well-recognized by researchers in the field. Schaie and Schaie (1977) doubt the adequacy of present psychological tests for the purpose of diagnosing cognitive impairment in the elderly. According to Schaie and Schaie (1977), general adult norms presently available cannot be applied properly to assess brain damage in the elderly. The performance of the elderly must be evaluated against the performance characteristic of the normal aging pattern. This is because some biological changes due to aging are inevitable and are therefore, likely to be reflected in cognitive functioning of the elderly. Thus, unless appropriate age norms are developed for the elderly, differential diagnosis cannot be made on the basis of existing psychological tests (Klisz, 1978).

Abnormal Aging Processes

Alzheimer's Disease

Alzheimer's disease (AD) is named after Alois Alzheimer who, in 1907, described a 51-year-old woman as suffering from a "peculiar disease of the cerebral cortex" (Slaby & Wyatt, 1974). The presenting symptoms of this patient were declining memory and disorientation. She became depressed and delusional over time. Within four and a half years, the patient became profoundly demented and showed severe impairment of speech and comprehension. The autopsy of her brain revealed atrophy, miliary lesions, and tangles or clumps of cortical neurofibrils.

Alzheimer's disease (AD) is an age-associated disorder involving atrophy of cortical tissues resulting in marked deterioration of intellectual and emotional functions. It is a progressive and irreversible disorder of the nervous system characterized by severe impairment of memory, cognition, and self-care ability. However, there are no visible physical symptoms, such as motor disorder and convulsions. Among the psychological problems, memory loss for recent events has been found to be the first and most prominent cognitive impairment in Alzheimer's disease (Brun, 1982, 1983; Folstein & Whitehouse, 1983; Hamill & Buell, 1982; McLachlan et al., 1984; Roth, 1980).

Alzheimer's dementia is characterized by deterioration in global cognitive functioning which includes memory, learning, orientation, and judgment. In addition, it is a disorder of language (aphasia), learned motor movement (apraxia), and visual recognition

(agnosia). These symptoms are also characteristic of mental retardation, amnesia, aphasia, and delirium. Mental retardation is distinguished from Alzheimer's in that the latter is a disturbance of acquired cognitive ability. Impairment of memory in Alzheimer's disease is different from amnestic syndrome, because other cognitive abilities in addition to memory are also impaired. It is also distinguishable from aphasic disorder due to the fact that other cognitive functions in addition to language are also affected. Alzheimer's disease is distinguishable from delirium, because delirium occurs under the condition of clear consciousness, whereas in dementia the victim is totally unaware (Schneck, Reisberg, Ferris, & Steven, 1982).

Although there is no fixed pattern of impairment over time, a progressive deterioration in linguistic ability and in the ability to read, write and perform mathematical operations is observed in Alzheimer's. It appears that the patient's ability to store information becomes deficient. As the disease progresses, the patient experiences difficulty in sentence formation and finding words that are appropriate for the context (Emery & Emery, 1983; Fuld, Katzman, Davies, & Terry, 1982). Manipulative and comprehensive language abilities are also impaired (Golper & Binder, 1981; Fuld et al., 1982; Martin & Fedio, 1983). Paraphasia and articulatory errors are also observed (Obler & Albert, 1980). Dysarthria and jumbling of sounds and words eventually interfere with the patients accomplishment of almost any intentional task, even intentional speech. In Alzheimer's patients, verbal retrieval capacity appears to deteriorate, while basic organization of language and syntax remains relatively intact

(Folstein & Whitehead, 1983; Hamill & Buell, 1982; Semple, Smith, & Swash, 1982).

Personality changes are evident in the disturbance of mood; the patients become labile, depressed, anxious, agitated and restless in the advanced stage of the disease. They are disoriented, confused, and experience delusions (Greer, 1982; Wells, 1977).

It is important to note that in the early stage, AD symptoms are similar to those observed in normal aging. But in later stages, AD is easily confused with other types of dementia and psychopathological conditions (Lauter, 1985; Reisberg, 1983).

Alzheimer's Disease and Senile Dementia

The literature on AD reveals that early researchers made a distinction between AD and senile dementia, perhaps because AD was considered a presenile disorder (before the age of 65). Dementia is a general term referring to "generalized cognitive and intellectual deterioration" (Reisberg, 1983). When this clinical syndrome is observed in patients older than 65 years of age, it is called senile dementia caused by any pathological process (e.g., tumor, multi-infarct, etc.). Some researchers use the term senile dementia of Alzheimer type (SDAT) to underscore the dichotomy of pre- and post-65 age in AD patients. However, most researchers do not find any significant difference between AD and SDAT (Gershon & Herman, 1982; Sourander & Sjorgren, 1970; Sulkava & Amberla, 1982).

From the morphological point of view, there are no significant differences between Alzheimer's disease and senile dementia (Sulkava & Amberla, 1982; Stornadt, 1973). In both conditions, the brain shows

cortical atrophy, widened cortical sulci, and enlarged ventricles. In addition, three histopathological changes are observed in both AD and senile dementia: (a) senile plaques, (b) neurofibrillary tangles, and (c) granulovacuolar degeneration of neurons (Lauter, 1985; Seltzer & Sherwin, 1983; Scheibel & Tomiyasu, 1978). However, these cortical changes are more abundant in Alzheimer's disease than senile dementia (McMenemey, 1970).

DSM III. For the purpose of diagnostic clarity, both terms - Alzheimer's disease and senile dementia - are discarded in DSM III (American Psychiatric Association, 1980). Instead, these two clinical entities are now placed under one category labelled Primary Degenerative Dementia (PDD). This category is further subclassified as (a) Primary Degenerative Dementia - senile onset, and (b) Primary Degenerative Dementia - presenile onset. According to DSM III, PDD refers to:

> ...a multifaceted loss of intellectual abilities, such as memory, judgment, abstract thought and other higher cortical functions and changes in personality and behavior (American Psychiatric Association, 1980, p. 124).

The following diagnostic criteria suggested by DSM III for Primary Degenerative Dementia incorporating Alzheimer's disease are: (a) dementia, (b) insidious onset with uniformly progressive deteriorating course, and (c) exclusion of all other specific causes of dementia by the history, physical examination, and laboratory tests. The third criterion suggests that differential diagnosis of dementia is extremely difficult, and that histopathological examination is presently the most reliable technique for accurate diagnosis of AD.

Course of the Disease

According to DSM III and other research reports, the course of Alzheimer's disease is uniform, gradual, and progressive (American Psychiatric Association, 1980; Reisberg, 1983; Schneck et al., 1982). Sjogren (1952) distinguished three stages in the development of this disease. The initial stage, which has been termed the "forgetfulness phase", lasts for two to four years. In this stage the deficits are primarily subjective. The patient shows slight cognitive and amnestic disturbance and spatial disorientation. There are subtle changes in the personality manifested through apathy, withdrawal, irritability, and lack of spontaneity. However, the patients in this early stage are cooperative and behave in a socially appropriate manner. They are able to continue working without any major hardship (Knesevich, Martin, Berg, & Danziger, 1983; Reisberg, 1983; Schneck et al., 1982; Sluss et al., 1980). Karl (1978) studied 94 subjects with a mean age of 80.5 for a period of four years. Forty subjects had clinically preserved memory functions, and twenty had senescent forgetfulness. Except in one case, no serious cognitive impairment was observed over the four-year period in these two groups. However, the remaining 34 subjects who were more severely impaired at the start of the study deteriorated rapidly and died earlier.

In the middle stage, termed "confusional phase", various cognitive deficiencies in AD become apparent along with changes in personality and behavior. Memory for recent events is quite impaired, but memory for past events remains intact (Schneck et al., 1982). Although vocabulary is not severely affected, the patients may experience difficulty in finding appropriate words. Observable

personality changes include denial and anxiety (Fuld et al., 1982; Greer, 1982). It is estimated that approximately three million Americans are presently suffering from this confusional symptomatology (Reisberg, 1983).

The third stage is called the "dementia phase". It is, in fact, the terminal stage. Deficits occur in all cognitive and functional areas. At this stage, cognitive changes are profound and there is marked disorientation of time, place and person. Motor activities are grossly impaired. The patient is unable to walk or grasp things by hands. He becomes inattentive, mute, incontinent and totally dependent upon others for self-care (Reisberg, 1983). In addition, there are severe personality disturbances. Psychotic symptoms, such as delusions, hallucinations, and paranoid ideation are explicit. The patient is also severely agitated and anxious (Lauter, 1985; Reisberg, 1983; Rosenstock, 1970). It is estimated that the dementia phase occurs in 5% of those aged 65 and over (Katzman, 1976; Terry & Davis, 1980). About 58% of the elderly patients in the U.S. nursing home suffer from the symptoms of the third stage (U.S. Dept. of Health, Education and Welfare, 1977).

Pathology of the Brain

Research on Alzheimer's has confirmed the earlier findings that there are distinct physiological changes in the brain. There is a general atrophy of the cerebral cortex which affects parietal, frontal, and temporal lobes (Brody, 1970, 1983; Terry, 1980; Tomlinson, 1982). Also, there are extensive pathological changes in the temporal-limbic structures (Brun, 1975, 1982, 1985; Wisniewski, 1983). The weight of the brain is greatly reduced (Brun, 1983;

Gottfries et al., 1983; Terry, 1980; Wisniewski, Terry, & Hirano, 1970). It has been hypothesized that loss of cortical neurons particularly in those ares responsible for memory, cognition and thought process is the cause of generalized atrophy of the brain in AD (Brody, 1970; Brun, 1982, 1983; Dekosky & Bass, 1982; Whitehouse et al., 1982). However, studies concerning neuronal loss differ in their findings. Shefer (1973) reports that Alzheimer patients have increased loss of neuronal cells in specific brain regions--median temporal, subiculum and hippocampus. Similar findings were reported by Brody (1983), Bondareff (1984), Bowen and Davison (1980), and Tomlinson (1982). These areas are primarily responsible for memory cognition and thought process (Brun, 1983, 1985; Terry, 1980). Brody (1983) and Wisniewski (1973) also found marked decrease in the number of neuronal cells in the hippocampus region of the AD patients. However, the research of Terry and associates (1977) did not confirm the above findings.

Another implication of the gross atrophy of the brain is that nerve cell extensions become atrophic due to loss of dendritic spines (Buell & Coleman, 1979; Scheibel, 1979, 1983). As a result, each neuron receives less information and the processing capability of the area involved diminishes (Brody, 1983). This produces progressive decline in sensorimotor, cognitive, and memory functions (DeKosky et al., 1982). In Alzheimer's disease capacity for neuronal growth is also reduced (Scheibel & Scheibel, 1975).

Microscopically, the major pathological changes in the brain of the Alzheimer's patient are in neurofibrillary tangles, senile plaques, and granulovacuolar bodies (Ball, 1983; Iqbal & Wisniewski,

1983). The neurofibrillary tangles (dense bundles of fibres within the nerve cell) are found mainly in the cerebral cortex, especially in the hippocampus area (Brun, 1983; Tomlinson, 1982; Wisniewski & Iqbal, 1980). Also, the neurofibrillary tangles replace nerve cells in the basal gangalia (Iqbal & Wisniewski, 1983; Tomlinson, 1982). Ball (1977) has shown that the posterior half of the hippocampus is much more affected by neurofibrillary tangles than the anterior half. Farmer & Associates (1976) found a positive correlation between a number of neurofibrillary tangles found in the posterior and anterior parts of the hippocampus and Alzheimer's disease.

Several studies have shown that neurofibrillary tangles are also present in the normally aging brain and in the brain of patients with other diseases (Matsuyama, 1983; Wisniewski et al., 1980). Matsuyama & Nakamura (1978) found that neurofibrillary tangles were present in the brains of more than 50% of the autopsied patients between age 50-59, in more than 80% of patients between age 60-69, and in 100% over age 70. It is also found in patients with dementia, Parkinson's disease and Down's syndrome (Wisniewski et al., 1979).

Senile plaques also called neuritic plaques is another microscopic lesion observed in the Alzheimer's brain (Iqbal, 1980; Reisberg, 1983; Tomlinson, 1982; Wisniewski, 1983). Senile plaques are mainly found in the cerebral cortex (frequently in temporal areas) and in the basal gangalia (Wisniewski & Iqbal, 1980; Wisniewski & Terry, 1973). A number of studies have shown that the degree of intellectual decline is associated with increasing number of senile plaques in the brain (Farmer, Peck, & Terry, 1976; Tomlinson et al., 1970). But senile plaques have also been found in normally aging

brains. In fact, they are more prevalent than neurofibrillary tangles in the brains of nondemented elderly (Winiewski & Iqbal, 1980). Matsuyama and Nakamura (1978), however, did not support this conclusion on the basis of their research.

The nerve cell lesion, known as "granulovacuolar degeneration" is also found in the hippocampus of the Alzheimer patients (Ball, 1983). The posterior half of the hippocampus is more severely affected. Ball and Lo (1977) studied the possible significance of granulovacuolar bodies in Alzheimer's disease. They reported that the patients with Alzheimer's disease have two to 100 times greater incidence of granulovacuolar degeneration than age-matched controls.

Although the etiology of Alzheimer's disease is still at the conjectural stage, there is no disagreement about the fact that the Alzheimer brain has serious pathology. In a recent paper, Khachturian (1985) has summarized the neuropathological lesions of AD patients as: (1) Neurofibrillary tangles, (2) Neuritic plaques, (3) Loss of neurons, and (4) Disturbance of acetycholine transmitter activity marked by lowered levels of acetylchonmine and choline acetyltransferase.

Incidence and Prevalence

A precise estimate of prevalence and incidence of Alzheimer's disease is difficult to obtain because of the lack of adequate diagnostic criteria. Prevalence refers to the number or percentage of the population suffering from the disease at a given time, whereas incidence refers to the occurrence of new cases in the population. In the United States, an estimated 2.5 million persons, mostly older than 65, suffer from this disease. Alzheimer's disease is the fourth

leading cause of death in America and accounts for 100,100 to 120,000 deaths per year (Katzman, 1976). Maletta, Pirozzolo, Thompson et al. (1982) report that about 69% of the elderly population suffer from this disease. In Canada, 10,000 persons die annually from this disease. It is estimated that about 100,000 to 300,000 Canadians are affected by AD to a varying degree. It is also the fourth leading cause of death in Canada (Health and Welfare Canada, 1984). In northern Europe, prevalence of severe dementia is 4-5% and prevalence of mild to moderate dementia is 11-12% (Katzman, 1976). Although about 4-5% of the 65 and over population suffer from severe dementia, the prevalence rate increases dramatically among the aged between 70 and 85 (Mortimer, 1983).

The incidence (percentage of new cases at a given time) of AD and senile dementia has not been studied extensively due to the problems associated with diagnostic criteria and logistics of monitoring the target population over a period of time. According to Mortimer (1983), a fair estimate of the annual incidence rate of senile dementia including AD is approximately 1% of the elderly population (over age 65). The incidence rate, however, levels off around the age of 75.

There is little information concerning the variations in prevalence of dementia by place, time and personal characteristics. However, it is clear that AD occurs in the adult population mainly, but strikes persons of all ethnic and socioeconomic backgrounds (Aronson, 1982). Although Kay (1964) reports that AD is more common among females, other studies do not support the notion of sex difference in the prevalence of Alzheimer's (Broe, 1976).

Nature of Cognitive Impairment

Since the primary characteristics of Alzheimer's disease are associated with cognitive functions, it is necessary to examine the nature of cognitive impairment for a better understanding of the disease. The following section will review briefly selected research on general intelligence, memory, and language functions of the paients suffering from senile and presenile dementia.

General Intelligence. Several studies have shown decline of general intellectual functioning in dementia (Miller, 1977). The results of the Wechsler-Bellevue or Wechsler Adult Intelligence Scale (WAIS) indicate that the verbal I.Q. is depressed in demented persons, while the Performance I.Q. is even lower. Cognitive decline has also been observed with Mill Hill Vocabulary Scale and Progressive Matrices (Kendrick, 1967; Masur & Fuld, 1982). A recent study of neuro-psychological patterns of presenile and senile dementia of the Alzheimer type revealed that Verbal, Performance and Full Scale I.Q. scores were significantly lower in the presenile group (Loring, 1985).

Some investigators attempted to examine the pattern of intellectual changes in Alzheimer dementia and normal aging. Robin (1945), Whitehead (1973), and Botwinick and Birren (1951) could not find any differences between the two groups. However, recent studies by Reisberg and his associates (1982a; 1982b) showed that although Alzheimer's patients did not differ from normals on WAIS in the early stage of the disease, there was a progressive decline in WAIS performance as the disease progressed. For a better understanding of

the pattern of intellectual decline in dementia, more research is needed.

Memory. Memory is one of the best researched cognitive aspects in dementia (Braddely, 1976; Craik, 1972; Ferris, Crook, & McCarthy, 1980). It has been found that memory and attention deficits account for much of the cognitive impairment in Alzheimer's disease (Vitaliano et al., 1986). Several studies have been conducted on short-term and long-term memory of demented persons. In the investigation of impairment of primary and secondary memory in dementia with Alzheimer types, Wilson, Bacon, Fox, & Kaszniak (1983) found that memory disorder of DAT was mainly the result of defect in primary memory. Miller (1977) in his study of short-term and long-term memory in dementia noted that demented patients were unable to repeat words immediately after single presentation. Short-term memory deficiency in dementia may be due to disturbance in the input system which gets the information into short-term memory (Miller, 1977). Another possibility might be an inefficient coding system utilized by the subjects for short-term memory.

There are very few studies dealing with long-term memory. In an experiment on long-term memory, Miller (1975) presented a list of words three times to demented subjects. The retention of the stimulus words was tested in three ways: (1) a straight forward recall test, (2) a recognition test, and (3) a partial information condition where the subjects were given initial letters of the words to be recalled. It was found that compared to the control group demented subjects performed very badly on recall and recognition tests. However, when the initial letters of the words were given, there was significant

improvement in the demented group. Miller (1975), and Warrington (1970) suggested that there might be retrieval difficulty or some other problem associated with long-term memory. In a recent study, Davis & Mumford (1984) investigated the retrieval deficit hypothesis of forgetting in senile dementia using a cued recall technique with eighteen AD patients and ten normal elderly. Their findings did not support a retrieval deficit of memory impairment in AD. Instead they suggest the possibility of a processing deficit at the acquisition stage. Encoding and processing deficits have been noted by Wilson, Bacon, Fox, & Kaszniak (1983) as contributing factors in impaired recognition memory of AD patients. However, there are very few studies regarding long-term retention and retrieval process of AD. More research on memory disorder is needed within the framework of the "levels of processing" model (Craik, 1977).

Weingartner et al. (1981) compared demented patients with depressed patients. They found that memory failure in patients with progressive dementia is different from that found in depressed patients. Demented patients had difficulty in seeing how events were related to one another in order to learn and remember. Depressed patients showed learning and memory failure when the tasks were difficult, whereas the patients with progressive dementia were unable to learn and remember even simple tasks. They also found that depressed patients' semantic memory was good, but that they had difficulty with episodic memory. However, the patients with progressive dementia were impaired in both semantic and episodic memory. Weingartner et al. (1983) in their study of episodic and semantic memory found that in AD patients both episodic and semantic

memory were grossly impaired. The progressively demented group had little access to previously acquired knowledge. Therefore, they had great difficulty in encoding and organizing the on-going events. In another study, impairment was found in both semantic and figural memory in the performance of AD patients on the Russell's Revised Wechsler Memory Scale (Brinkman, Largen, Gerganoff, & Pomara, 1983). Nebes, Martin, and Horn (1984) reported severe semantic memory deficit in AD patients.

A thorough study was conducted by Corkin (1981) to see whether impaired learning and memory manifested by amnesic patients were similar to the memory loss found in Alzheimer's disease. A group of healthy elderly subjects were also included. Short-term memory was measured by digit span, block span, and the Brown-Peterson distraction paradigm. The study found that there was no difference between the healthy and amnesic elderly subjects on digit span and block span, but the moderately and severely demented groups were significantly lower. The mildly demented group fell in the middle range. Similar findings were reported on the Brown-Peterson Distractor Scale. The encoding process among these groups was assessed by the processing framework of Craik & Lackhart (1972). An attempt was made to assess three levels of processings: (a) sensory, (b) phonological, and (c) semantic. It was found that Alzheimer groups performed poorly in all three types of questions.

Language. Although memory has received the attention of most researchers, there are a few studies on the nature of language impairment in dementia. Ernst, Dalby, and Dalby (1970) did a systematic examination of language functioning in demented patients.

They found that there was general poverty of vocabulary in the narrative speech. They also misnamed objects more often than the control group. It was suggested that misnaming of objects among demented patients is due to misidentification of the objects (Baker, 1968; Rochford, 1971). Language disorder and apraxia were reported by Brun et al. (1984) in 78% of the AD cases.

Multi-Infarct Dementia (MID)

Another commonly observed dementia among the elderly is Multi-infarct Dementia (MID). It is a vascular disorder of the brain responsible for mental decline via small or large cerebral infarcts (Hachinski, Lassen, & Marshall, 1974; Hachinski, 1983). The prevailing view is that the atherosclerosis of the cerebral vessels causes critical narrowing, leading to chronic shortage of blood to the brain which, in turn, contributes to the mental deterioration (Hachinski, 1983; St. Clair, & Whalley, 1983). The middle cerebral artery on the left side is the most commonly reported site of such disorders. Arterosclerosis is the main underlying cause of cerebral infarction that causes focal rather than global intellectual impairment (Wells, 1977, 1980). The immediate pathological process consists of multiple small strokes that eventually lead to diffuse softening and degeneration of cerebral tissues (Scheinberg, 1978; Walton, 1977). This condition occurs when 50-100g of brain tissues have been damaged due to loss of elasticity ~~with~~ thickening of arterial walls (Torack, 1978; Walton, 1977). At the time of autopsy, multifocal damage to the brain is usually visible to the naked eye (Hachinski, 1983).

Alzheimer's Disease vs. Multi-Infarct Dementia

Although behavioral symptoms of Multi-infarct Dementia are generally indistinguishable from Alzheimer's disease (Torack, 1978), MID tends to have a number of distinguishing features (Hachinski et al., 1974; Roth, 1978, 1980; Walton, 1977). In Multi-infarct Dementia, the onset may be abrupt and the deterioration stepwise accompanied by focal neurological signs (Marshall, 1972). The course of illness is generally intermittent, resulting in episodes of marked cognitive deficits. The pattern of cognitive deficits is patchy depending upon the regions of the brain involved (Butler & Lewis, 1982; Loeb, 1980). The severity of symptoms may fluctuate from hour to hour and day to night with nocturnal confusion. At first, personality is less affected in MID than in AD. However, the later stages of MID reveal severely distorted personality and cognitive changes (Hachinski, 1983; Roth, 1980).

Memory impairment of recent events is one of the earliest signs in all types of dementia, but in Multi-infarct Dementia, onset is gradual and spotty (Butler & Lewis, 1982). Perhaps the most distinctive characteristic of MID is motor abnormality, gait disturbance and extreme rigidity (Scheinberg, 1978). This suggests the involvement of subcortical structures of the brain. Also, patients with cerebral infarction show great emotional variability, from being indifferent to being unduly emotionally dependent, depending upon the site of the lesions. Severe anxiety and depression are also more common and prominent in MID than is the case with AD patients. This may be a reaction to the accurate perception of their failing mental abilities (Liston & LaRue, 1983; Loeb, 1980; O'Brien,

1977). Death usually occurs within five years (Hachinski, 1983).

Although the etiology and course of Multi-infarct Dementia are believed to be different from Alzheimer's disease, there are a number of common symptoms (Sluss, Robin, Gruenberg, & Kramer, 1982). For example, dysphasia (difficulty in speaking or understanding speech) and aphasia (total loss of articulated speech) are often present in both types of dementia. Impairment of intellect and recent memory is also found in both dementias. Other common characteristics include impulsiveness, mood disturbance and personality disorder (Pitt, 1982; Predesu, Alexianu, Tudorache & Tudor, 1983).

In spite of the above-mentioned shared characteristics, there are several focal neurological signs and symptoms which are useful in differentiating AD from MID. Visual defects, ataxia (loss in muscle coordination affecting gait and posture), slurring of speech, and dysphasia are typical in MID. Transient ischemic attacks, long standing hypertension, dizziness, somatic complaints, a history of strokes and artherosclerosis generally accompany MID (Ladurner & Lencher, 1982; Sluss et al., 1982; Wells, 1977). From the clinical point of view, it is crucial to make a distinction between Multi-infarct Dementia and other forms of dementia, particularly AD, since MID may be a treatable condition. Appropriate treatment is likely to not only arrest progression of MID, but may produce some recovery from the disease (Hachinski, 1983; Liston & LaRue, 1983).

Incidence and Prevalence

Multi-infarct Dementia is somewhat less prevalent than Alzheimer's type dementia. On the basis of a survey study (Tomlinson, 1970), it was estimated that approximately 20% of all cases of

dementia were clearly of Multi-infarct nature, whereas 13% of cases were identified as mixed dementia exhibiting both Alzheimer's type changes as well as diffuse cerebral softening. In a more recent study, Maletta and Pirazzola (1982) report that about 10% of the elderly population suffer from Multi-infarct Dementia. The ratio of primary degenerative dementia to Multi-infarct Dementia is about four to one. Multi-infarct Dementia is more common in individuals between the ages of 50 to 70 and is more frequently seen in men with a history of hypertension (Butler & Lewis, 1982; Hagnell et al., 1983; Ladurner & Lencher, 1982).

Depression

With respect to the nature of psychopathology in old age, Butler's (1975) review of the literature suggests that the rate of psychoses, functional disorders (especially depression and paranoid states), as well as organic brain disorder increase steadily after the age of 60. A large number of studies have indicated that the most prevalent serious emotional disorder in old age is depression (Butler & Lewis, 1982; Finlayson & Martin, 1982; Magni, De Leo & Schifano, 1985). It is generally believed that social and personal circumstances in old age are conducive to depressive illness. Researchers as well as clinicians suggest that depression in the elderly is more complex, confusing and difficult to diagnose. This is so because a multitude of factors--physical, social, psychological and environmental--affect old persons simultaneously (Murrel, Himmelfarb & Wright, 1983; Roth, 1976; Straker, 1984). At present, there are very few studies comparing the symptoms of older and younger depressive

groups. Although the findings are not conclusive, it is generally argued that late life depression follows a somewhat different pattern than that commonly observed in the younger age groups (Perlick & Atkins, 1984; Raskin & Rai, 1980).

Characteristics of Depression in Old Age

As for symptoms of depression in the aged, there are little empirical data that accompany clinical observations. Based on a comprehensive review of the literature, Raskin and Jarvik (1979) have prepared a list of major signs and symptoms of depression in old persons. These include disturbance of memory, attention or concentration, sleep and appetite. Other signs include dysphoric mood, pessimism and inadequacy, apathy and social withdrawal, confusion or perplexity, and somatic complaints. Furthermore, loss of self-esteem, inferiority feelings, listlessness, sense of helplessness, crying, irritability, infantile behavior, self-deprecation, agitation, and excessive feelings of guilt and anxiety are also common characteristics of old age depression (Butler & Lewis, 1982; Salzman & Shader, 1978; Winokur, Beham, & Schlesser, 1980).

Presence of excessive guilt among the depressive elderly is a controversial observation (Blazer, 1982). Butler & Lewis (1982) believe that guilt is an important aspect of depression among the elderly who are likely to feel guilty over many past events of their life which they regret. But Pfeiffer and Busse (1972) reported that guilt is not as important as self-regard in the depressive elderly. However, several studies reported that guilt is either not present or less prevalent in the depressed elderly (Blazer, 1982; Winokur, Behan,

& Schlesser, 1980).

Somatic complaints of the elderly have been associated with depression. The elderly are more likely to report their physical complaints than their feelings of depression (Blumenthall, 1975; Butler, 1982; Murphy, 1983; Zung & Green, 1972). Commonly reported somatic complaints by the depressive elderly include insomnia, loss of appetite, fatigue, headaches, constipation and various other gastrointestinal problems (Butler & Lewis, 1982; Gurland, 1976). However, Salzman and Shader (1975) pointed out that it is difficult to associate somatic complaints such as constipation and fatigue with depression, because they are often the result of physical decline with advancing age rather than depression.

We cannot assume that complaints of memory loss and difficulty with concentration and attention, which are often reported by the depressed elderly, are simply consequences of depression, since many physical and neurological diseases associated with aging also accompany loss of memory and inability to attend and concentrate. Accurate diagnosis of depression is essential, because memory loss commonly present in depression is likely to respond to treatment (Folstein & McHugh, 1978; Gibson, 1981; Kahn, Zarit, Hilbert, & Niederehe, 1975; Plorkin, Mintz, & Jarvik, 1985). Anxiety is another frequently reported symptom among the depressed elderly that is also present in dementia. Eisdorfer, Cohen, and Keckich (1983) have demonstrated that anxiety is more commonly associated with depression than dementia.

Incidence and Prevalence of Depression in Old Age

Lack of sufficient research and clear criteria for depression

in general and particularly depression in late life do not allow accurate estimates of the incidence and prevalence of depression in the elderly. Also, where and how the studies have been conducted influence the calculation of actual incidence and prevalence (Zarit, 1980). In general, the rate of depression reported in the aged is high. According to Dovenmuehle, Reckless, and Neuman (1970), approximately 33% of 60-year-olds and over suffer from some form of depression. As for the specific type of depression, several studies have shown that psychotic depression is more frequently diagnosed in those 60 and over. Also, it is estimated that at the time of admission, between 21% to 54% of geriatric patients receive the diagnosis of psychotic depression (Gurland et al., 1980; Pfeiffer & Busse, 1973). According to Straker (1984), two out of every three elderly patients seeking psychiatric attention suffer from a depressive illness. Blazer (1982) reported a 33% incidence of depressive diagnosis among older patients. Redlick & Taube (1980) in their evaluation of various psychiatric facilities in U.S.A. found that 38.4% were diagnosed as depressives. They also noted that depressive diagnosis varied in different institutional settings.

A higher prevalence of depression has also been noted in surveys of nonpatient older populations. It was estimated that between 2% and 10% of the elderly living in the community were depressed enough to receive a clinical diagnosis of depression (Gurland, 1980). A considerably higher prevalence was reported in another community sample of persons over age 65. Harwin et al. (1973) observed that about 50% of the elderly had either mild or severe depression. Depression is the most common complaint among healthy

community residents (Redlich & Taube, 1980).

Cognitive Changes in Elderly Depressives

Some forms of cognitive deficit in old age depression have been clearly recognized, specially those concerned with attention, orientation and memory (Eisdorfer, Cohen, & Keckich, 1983). However, there is a great deal of controversy among researchers about the nature and extent of cognitive deficit in the elderly. In recent years, the research focus has been in the areas of memory and learning (Breen et al., 1984; Murphy, 1983; Okimoto, Barnes, Veith, Raskind, Inul, & Carter, 1982; Plotkin, Mintz, & Jarvik, 1985; Post, 1982). However, there are very few studies that have utilized psychological tests for understanding the nature of brain functioning in old age depression. Savage, Britton, Bolton, and Hall (1973) examined the WAIS performance of hospitalized depressed patients and found that they scored lower on both performance and verbal subtests compared to normals. Similar findings were reported by Nunn, Bergmann, and & Britton (1974). Also, Donnelly, Murphy, Goodwin, and Waldman (1982) did not find any decline in intellectual functioning of a group of hospitalized elderly depressed patients. In contrast, Whitehead (1973) in evaluating the WAIS performance of the elderly concluded that the pattern of deficits is the same in the normal, depressed and demented elderly.

With respect to memory and learning, some contradictory findings have been reported. In terms of primary memory, Whitehead (1973) concluded that depressives were not impaired on the Digit Span subtest of the WAIS. But these findings are questionable, since the elderly subjects included in his study were only those with dementia

and depression--without a control group of normal elderly. In contrast, Savage et al. (1973) using appropriate controls observed impairment on the digit span scores of the aged. Whitehead (1973) extended the same study and found that depressives did poorly on the Synonym Learning Test and the Serial Learning Test. They were also impaired on paired-associate learning, memory for passages, and recognition tasks. Similar findings were reported by Irvings, Robinson, and McAdam (1970). Davices, Hamilton, and Hendrickson (1978) support the earlier findings that depressed aged do poorly on paired-associate and serial learning tasks. On the Modified Learning Test, Savage et al. (1973) noted that the affective group did better than the dements, but still performed below normals.

Psychomotor retardation in the depressed elderly has generally been confirmed by research findings (Neville & Folstein, 1979; Gibson & Moyes, 1979). However, Friedman (1964) in his study of 55 depressed and 65 normal subjects failed to find any significant differences between the two groups with regard to psychomotor performance. According to Wood, Ebert, and Kinsbourne (1982), the depressed elderly may have mild, reversible motor impairment.

Differential Diagnoses of Alzheimer's Disease, Multi-infarct Dementia and Geriatric Depression

Notwithstanding the fact that Alzheimer's disease has been studied by clinicians since 1910, scientific knowledge about its nature, etiology, diagnosis, prognosis and prevention is still very inadequate. As mentioned before, the most distinguishing feature of

AD is progressive deterioration of cognitive functions including intelligence, memory and judgment. However, these symptoms are also observed in several other disease conditions, such as Huntington's chorea, brain tumor, Multi-infarct Dementia, etc. The clinical picture is further complicated by the fact that AD is often accompanied by delirium, delusion and depression creating various subtypes of the disease as suggested in DSM III (American Psychiatric Association, 1980).

Depressive symptoms can be manifested in many ways and often are continuous with the organic symptoms of senile dementia. Pseudodementia is the clinical term for the syndrome in which depression masks as dementia. Typical symptoms include confusion, disorientation, poor concentration, slowness in responsivity, deficits in short- and long-term memory, and diminished conceptual abilities (Janowsky, 1982).

Barry Reisberg (1983) has compiled a comprehensive list of clinical features in selected disease conditions with dementia. The following table (Table 2) discusses the onset and course of cognitive impairment in the three diagnostic groups.

Assessment of Dementia

Physiological Methods

Accurate diagnosis of Alzheimer's disease is the major problem encountered by both researchers and clinicians working in this field (Filinson, 1984). Presently, the only reliable procedure is an autopsy or biopsy which allows for microscopic examination of brain

The material presented on Page 52 has been removed because of the unavailability of copyright permission.

This page contains Table 2 showing major clinical features of Alzheimer's Disease, Multi-infarct Dementia and Depression.

The original source of the information presented in Table 2 is Reisberg (1983, p.184).

tissues to determine the presence of amyloid plaques and neurofibrillary tangles. There is no other psychological or physiological procedure that is capable of providing definitive diagnosis of AD during the lifetime of the patient. As a consequence, clinical diagnosis of AD is essentially a diagnosis of exclusion (Crook & Miller, 1985; Kushner, 1982; Roth, 1978).

The present situation of diagnostic uncertainty is quite disturbing. The percentage of incorrect diagnoses is alarming, it ranges from 10-30% in the general medical population (National Institute of Aging Task Force, 1980). Garcia, Reding, and Blass (1981) report 10% to 50% error rate in distinguishing early dementia from depression. Misdiagnosis of AD is frequent during early stages of the disease. This creates difficulty in developing appropriate treatment and management plans. Accurate diagnosis is extremely important for behavioral research on AD so that the disease could be studied systematically from early to later stages.

Clinical diagnosis of AD is unclear and complicated, since several other physiological and psychological disorders have similar symptoms. For example, Pick's disease, Multi-infarct Dementia, depression, pseudodementia, alcohol abuse, heavy metal poisoning, nutritional deficiencies, infectious disease, trauma, and circulatory disturbance share dementia as the common pathological symptom. However, in many of these disorders, the apparent dementia can be reversed if identified accurately and treated in the early stage (Hachinski, 1983; Khachturian, 1985; Reisberg, 1983; Schneck, 1982; Wells, 1983).

A variety of physiological and chemical techniques have been

utilized during the past 10-15 years by researchers and clinicians, primarily for the purpose of screening patients with reversible dementia (Lauter, 1985; Wells, 1983). Recent technological advances have made it possible to obtain noninvasive imaging of the brain. These techniques provide much more accurate information about neuroanatomical aspects of organic disorders. Computerized tomography (CT), single photon emission computed tomography (SPECT), and nuclear magnetic resonance (NMR) are the major neuroimaging methods at the present time (Khachturian, 1985). Several laboratory diagnostic techniques have also been found to be useful in differential diagnosis of dementia (Wong, 1980), such as measure of regional cerebral blood flow (rCBF) (Hachinski et al., 1975; Yamaguchi, Meyer, & Yamamoto, 1980), Electroencephalography (EEG), a most widely used and easily available technique in most medical facilities (Busse, 1980), average evoked potential (EP), an electrophysiological technique (Hendrickson et al., 1979), and brain biopsy, an electromicroscopic examination of the brain tissue.

In order to rule out reversible causes of dementia and cognitive impairment, several auxiliary tests have been suggested by Wells (1979). They are: serum enzymes and electrolyes, complete blood cell count, urine analysis, chest X-ray, serological test for syphilis, serum thyroxine, thyroid tumor tests, vitamin B_{12} and folate levels. These tests in conjunction with other major physiological techniques are likely to improve efficiency in diagnosing degenerative brain disorder. However, it should be noted that even highly sophisticated neurophysiological techniques are not completely reliable thereby providing considerable false positives, particularly

in the case of Alzheimer's disease.

Psychological Methods

Although psychometric instruments have been employed to assess higher nervous system disorders since the time of Binet and Simon (circa 1900) in France, behavioral effects of specified brain lesions manifested in test performance began to be studied systematically only during the 1930s and 1940s. In their excellent review of the field, Jones and Butter (1983) refer to a number of early studies where selected psychological tests were used to study specified problems, such as intellectual impairment in aphasics, memory impairments in amnesia, and the effect of brain lesions on intelligence and abstraction abilities. These studies were conspicuous by their use of highly specialized psychological tasks within the framework of traditional psychometric methods. In this connection, Luria's contributions must be acknowledged. He started experimental-clinical work in the 1930s and continued until 1977 when he died (Luria, 1980). His major contributions were compilation of a variety of psychological tests for clinical use and theory building about brain-behavior relationships.

Psychological methods focus upon the individual's performance on a variety of tasks that are sensitive to neuropathological conditions. It is now acknowledged that psychological instruments provide a comprehensive picture of cognitive impairment (Goldstein, 1984; Lezak, 1983). The psychological test batteries are not only objective compared to clinical evaluations (Kahn, 1975), but they also provide a relatively precise assessment of the severity of cognitive

deficits in Alzheimer's and other disorders. Such knowledge is essential for developing appropriate diagnosis, treatment, management and rehabilitiation plans for the patients.

Presently, a number of psychological tests are used for the diagnosis of dementia and depression. Among the commonly used instruments are: Wechsler Adult Intelligence Scale (WAIS), Wechsler Memory Scale (WMS), Raven's Progressive Matrices, Fuld's Object-Memory Evaluation Test, Halstead-Reitan Battery, Aphasia Screening Tests, Trail Making Test, Wisconsin Card Sorting Test, and Luria-Nebraska Neuropsychological Battery. Recently, Hachinski (1983) has identified thirteen items to derive an Ischemic score to assess dementia. Hachinski's Ischemic score has been validated by several researchers as an effective clinical tool for differential diagnosis of AD and MID (Reisberg, 1983). However, the precise nature of neuropsychological impairment cannot be assessed with the Ischemic score, since it is based primarily upon clinical observations.

Hemisphere Functions of the Brain

Since neuropsychological methods acknowledge the importance of hemispheric specializaiton in cognitive functioning, the following section will present a brief reveiw of cerebral lateralization and its behavioral implications.

The cerebral cortex, which is the most highly organized correlation centre of the brain, is divided into two morphologically identical, but functionally different parts (Golden, 1978; Lezak, 1983). Both the right and the left hemispheres are considered

responsible for higher mental functions. 'Cerebral Lateralization' refers to the anatomic and functional differences between the two halves of the brain. They differ in primary cognitive functioning and in processing behavior (Kinsbourne, 1982). The left is specialized for the analytical, logical mode in which verbal aspects are more involved, whereas the right is specialized for the holistic, gestalt mode which is concerned with visio-spatial relations. Thus, the main functional difference between the right and the left hemisphere is that the left hemisphere in most people is dominant for speech or verbal stimuli such as reading, writing, understanding and speaking, whereas the right hemisphere is mainly concerned with mediating complex nonverbal stimuli. Also, right and left hemispheres differ from each other in basic processing of the stimuli. The left hemisphere has been considered by Nebes (1974) as the 'analyzer' and the right hemisphere as the 'synthesizer'. De Renzi and Faglione (1967) have hypothesized that functions of the right hemisphere are more diffuse in nature whereas functions of the left hemisphere are more focal. Similar findings were reported by Semmes (1968). She found that right hemisphere damage produces tactile impairment in both hands, while persons with left hemisphere damage experienced tactile impairment only in the contralateral hand. Kertesz and Dobrowolski (1981) also support diffuse organization of the right hemisphere.

The two hemispheres differ not only in their cognitive activities, but also in their emotional behavior. Different emotional changes are noted in injury to left and right hemispheres (Sackeim et al., 1982; Valenstein & Heilman, 1979). Patients with right hemisphere impairment feel euphoric and are less likely to be aware of

their mistakes, thereby behaving inappropriately in social situations. Emotional tone, facial expression and quality of voice are all under the control of the right hemisphere. Thus, the person with right hemisphere dysfunction experiences difficulty in showing appropriate emotions (Morrow, Vrtunski, & Boller, 1981). The common emotional behaviors noted in left hemisphere dysfunctions are anxiety and depression. Catastrophic reaction and agitation are also present (Gainotti, Cianchetti, & Tiacci, 1972; Galin, 1974). However, it should be noted that it is extremely difficult to separate the functioning of the two hemispheres, since they are tightly connected together by the corpus collosum (Golden,1979; Lezak, 1982c).

In the following section functional characteristics of both hemispheres will be reviewed separately.

Left Hemisphere

As mentioned earlier the left hemisphere is primarily responsible for verbal functioning. It involves reading, writing, speaking, verbal memory and comprehension of verbal materials. Thus it is more expressive in nature (Lezak, 1982). Patients with left hemisphere dysfunction are likely to have difficulty in understanding and articulating speech because their ability to break down words into basic phonemes is impaired (Luria, 1966). They are unable to produce sentences in logical, grammatically correct and understandable manner. Ability to solve mathematical problems is also impaired (Luria, 1966). Difficulties in remembering verbal materials and speaking words correctly are other noticeable impairments (Luria, 1966). According to Luria (1966), the verbal type of spatial ability is dependent on

the left hemisphere. A lesion in the left hemisphere affects the ability to understand spatial relationship, especially when verbal command is required to complete the tasks. The patients also have difficulty in understanding words that convey spatial relations, such as the words below and above (Benton, Levin, & Van Allen, 1974; Luria, 1966).

Right Hemisphere

The right hemisphere is primarily nonverbal with limited capacity for verbal material (Zaidel, 1978). The right hemisphere processes information through visio-spatial modalities. Damage to the right hemisphere affects the ability for tactile and visual recognition, visiospatial memory, drawing, remembering nonverbal material, ability for spatial orientation, discrimination of colors according to hue and discriminating musical sounds (Lezak, 1982; Searleman, 1977). Poor rhythm of speech and tone quality are evident in the monotonous speech that is characteristic of the right hemisphere impairment. Automatic functions are also impaired. Heilman and Van Dee Abell (1980) reported that reaction time mediated by the right hemisphere is faster than that of the left hemisphere. Thus, the right hemisphere can be considered dominant in attention.

Considerable research has been done to determine the role of the right hemisphere in spatial orientation and awareness. It has been consistently noted that the right hemisphere is important in depth perception and stereoscopic vision (Lezak, 1982). The ability to perceive two or three dimensional relationships is affected by the right hemisphere lesion (Durnford & Kimura, 1971). Visiospatial

memory, perceptual conceptual organization, and spatial orientation are also defective due to the right hemisphere impairment. Patients have difficulty in finding their ways around (Carman & Nachshon, 1971; Dee & Van Allen, 1971). Patients with the right hemisphere lesion experience difficulty in sequencing, ordering, and making sense out of complex situations and stimuli when verbally presented (Lezak, 1982). However, some verbal abilities, primarily receptive in nature, involve functioning of the right hemisphere (Gott, 1973).

Functional Units Within the Hemispheres

Each hemisphere is divided into four lobes: (a) Parietal lobe, (b) Frontal lobe, (c) Occipital lobe, and (d) Temporal lobe. A brief review of the functions of each is presented below.

Parietal lobe

The main function of the parietal lobe is to process somato-sensory information from the body (Luria, 1965, 1973). The pariental lobe contains secondary and tertiary association areas. Damage to these association areas may produce impaired recognition of various stimuli (Lezak, 1983). Inability to recognize objects or discriminate between two points by touch (astereognosis) is prominent in the parietal lobe disturbance. Finger agnosis, the inability to rocognize which finger is touched, may also result due to damage to either side of the parietal lobe (Gainotti, Gianchetti, & Tiacci, 1972).

The impairment of parietal-occipital area is involved in the ability to understand and manipulate arithmetic symbols (Benson & Weiss, 1972). However, deficit in this area does not affect the ability to do well-memorized arithmetic problems where only memory is

involved, nor the ability to solve mathematical problems. This impairment also affects the ability to relate verbal and spatial concepts. For example, the patient with parietal-occipital damage is unable to tell time only by the spatial position of the hands of the clock. The understanding of words which indicate spatial relationships, such as above and below is likely to be impaired (Luria, 1973; Lezak, 1983).

The parieto-occipitotemporal area is responsible for integrating information from all sensory madalities and is important in many speech processes. Reading and writing skills, and the ability to associate names with objects are adversely affected by damage to this area (Butters & Brody, 1969; Luria, 1966). Also, deficit in verbal memory results from lesion in this area (Warrington & Robin, 1970).

Frontal lobe

The frontal lobe, the largest part of the cerebrum, is responsible for all intellectual activities, such as the ability to think, use language, and utilize the higher mental processes. Cognitive and emotional behaviors are mediated through the frontal lobe. It is the site of interconnections and feedback between major sensory and motor systems of the body. The frontal lobe correlates incoming information from all sources--external, internal, conscious, and unconscious (Luria, 1973). According to Luria (1973) frontal lobe disturbance tends to have repercussions throughout the behavioral repertoire.

Very briefly, some specific cognitive defects of frontal lobe lesions are: (a) problems with learning new associations with

previously learned stimuli (Milner, 1971), (b) defective abstract thinking and perseveration and rigidity in response (Lezak, 1983), (c) association between language, behavior and ongoing activities, (d) defective visual scanning ability (Teuber, 1964), (e) impaired short-term memory (Lewinsohn, Zieler, Libert, Eyberg, & Nielsen, 1972), (f) lack of imaginative and innovative thinking (Zangwill, 1966), and (g) impaired orientation and judgment of time (Benton, 1968).

Occipital lobe

Lesions in the visual association area of the occipital lobe result in visual agnosia or visual distortions (Lezak, 1983). In visual agnosia the patient is unable to synthesize input into a pattern form. General intellectual deterioration is also noted (Rubens, 1979). Disturbances in the occipital lobes also produce simultaneous agnosia, where the patient has difficulty in perceiving two objects or two sensory attributes of a single object at the same time. Since objects are not seen as a whole, the person has difficulty in recognizing the objects. Luria (1965) has attributed this to the problem in shifting attention stemming from disorder of the motor movement of the eyes. This causes difficulty in scanning or providing direct attention to important details of the stimulus.

The patients with left occipital damage may have difficulty with reading or recognizing the letters or numbers (Alberta, Reches, & Silverberg, 1975; Damasio, 1977). They are also unable to remember verbal material presented in visual form (Benson, 1977). Damage to the right occipital areas is likely to cause disturbance in spatial-perceptual orientation to visual stimuli (De Renzi, Faglioni, & Scott,

1970). As a result, a person may have difficulty in localizing oneself in space by means of visual cues (Oxbury, Campbell, & Oxbury, 1974).

Temporal lobe

The temporal lobe is mainly concerned with hearing and related functions, such as auditory memory, and complex perceptual organization. The anterior-medial area of each temporal lobe is closely associated with the limbic system. As a result, lesions in the temporal lobe are likely to produce visual or auditory hallucinations which may lead to personality changes and a variety of psychiatric problems usually associated with limbic disorders (Luria, 1966). Learning as well as long-term memory are affected by the loss of those areas found in hippocampal disorder. Sequencing ability is also impaired by damage to the temporal lobe (Milberg, Cummings, Goodglass, & Kaplan, 1979). However, general intellectual ability is not affected (Milner, 1972).

Left temporal lobe lesions are likely to disrupt receptive language processes. They also affect the ability of phonetic analysis necessary for reading, writing and speaking. The patient is unable to monitor his or her own speech (Fedio & Van Buren, 1975). The patients with lesion in the right temporal lobe have problems with nonverbal sound comprehension, discrimination and recognition. Their ability to distinguish tones, tonal patterns and beats are impaired (Luria, 1963; Vignolo, 1969).

Neuropsychological Assessment

Neuropsychology and Clinical Neuropsychology

Neuropsychology is a rapidly developing branch of psychology, although it started as a subspeciality of clinical psychology in the United States shortly after World War II (Matarazzo, 1972). It is an applied science, based upon the basic disciplines of neurology and psychology, and is concerned mainly with the study of brain-behavior relationships. According to Hamsher (1983), neuropsychology is the study of functional output of the central nervous system. Because of its emphasis on brain-behavior relationship, the field of neuropsychology has become central to a variety of basic and clinical disciplines, such as medicine, neurology, psychiatry, rehabilitation medicine, gerontology, etc. In recent years, increasing use of neuropsychological knowledge is being made in education, employment, and forensic work.

A sub-specialization of neuropsychology is clinical neuropsychology where the primary focus is upon behavioral expression of brain dysfunction. Information about etiology, severity, and localization of cerebral impairment is essential for identifying neurobehavioral syndromes and for making accurate diagnoses (Hamsher, 1983). Clinical neuropsychologists not only help in the diagnosis of organicity, but they also provide expertise in developing appropriate plans for treatment, management, and rehabilitation (Goldstein, 1984; Hamsher, 1983; Lezak, 1983).

Rationale and Goals of Neuropsychological Assessment

Clinical psychologists have always been interested in the assessment of brain damage or organicity, since abnormal test performance may be due to psychopathology, mental retardation or organic involvement. In recent years, the term neuropsychological assessment has replaced the old terminology--"tests of brain damage or organicity"--to reflect the advanced state of knowledge of neuropsychology. The basic goal of neuropsychological testing is assessment of brain functions through behavior. Ralph Reitan (1974) explains that neuropsychological assessment consists of tests that are "sensitive to the conditions of the brain" so that change in brain functions is reflected in test performance.

It is well-known that injury to different regions of the brain produces different behavioral changes. By examining particular behavior changes, it is possible to predict the site and size of brain impairment. According to Goldstein, the aim of neuropsychological assessment is "...largely that of determining the pattern of the patient's preserved and impaired functions and inferring from this pattern what the nature might be of the disturbed brain functions" (1984, p.186). The foremost goal of neuropsychological assessment is to provide an accurate diagnosis of brain dysfunctions. According to Lezak (1983) clinical neuropsychological evaluation must have three main goals: (a) diagnosis, (b) patient care or rehabilitation, and (c) research.

Thus, neuropsychological assessment is essentially an evaluation of the brain's capacity for rational thought, communication, memory, learning, perception, motor functions, etc

(Hamsher, 1983). As is obvious, such an assessment should be extremely useful in differential diagnosis of psychiatric as well as organic disorders.

As discussed before, there are several good physiological measures such as CT scan, PET scan, MNR, etc., that have been employed in determining the site and size of brain lesions. Yet no assessment of brain damage is complete without a comprehensive assessment of neuropsychological functioning (Garcia, Reding, & Blass, 1981). In fact, the demand for neuropsychological assessment has been growing steadily in the past ten years among mental health professionals and behavioral scientists.

Comprehensive Neuropsychological Assessment

At present, there are two major clinical approaches to neuropsychological testing. One is a specialized approach in which tests are selected individually for each patient on the basis of referral questions, clinical interview, and history of patient. This approach is called the "flexible approach" (Benton, 1977). Another is a comprehensive battery approach in which a fixed set of neuropsychological tests are administered. A comprehensive neuropsychological assessment battery is defined by Goldstein (1984) as"...a procedure that assesses all of the major functional areas generally affected by structural brain damage" (p.181). The major functional areas include cognition, perception, attention and motor skills. Thus, a comprehensive neuropsychological test battery is supposed to be able to cover all areas important to the understanding of brain-behavior relationships. Goldstein (1984) explains

comprehensive neuropsychological assessment as follows:

> "...assessment typically involves the functional areas of general intellectual capacity, memory, speed and accuracy of psychomotor activity; visual-spatial skills; visual, auditory and tactile perception; language and attention. Thus, a comprehensive neuropsychological assessment may be defined as a procedure that at least surveys all of these areas. In practical terms, a survey is all that is feasible if the intent of the assessment is to evaluate all areas..."(p.185).

Although several individual tests are available and are being widely used for screening organicity, at present there are only two main comprehensive neuropsychological test batteries, namely the Halstead-Reitan Battery and the Luria-Nebraska Neuropsychological Battery. Both these batteries share a common purpose, but they differ in several important ways. A brief description of these two instruments is likely to provide better understanding of the nature of comprehensive neuropsychological tests.

The Halstead Reitan Battery

Since its development in the late 1940s, the Halstead-Reitan Battery has been widely used in clinical settings. Several modifications have been introduced in this battery on the basis of clinical and empirical research during the past 40 years (Golden, 1978; Reitan & Davison, 1974). The test battery was developed by Reitan who expanded the earlier laboratory work of Halstead in 1935 with neurological patients. Since content of the test battery is somewhat flexible, clinicians and researchers can select a number of tests covering a wide range of functional areas. However, most clinicians use a set of 11 tests. They are: (a) Wechsler Adult Intelligence Scale (WAIS), (b) Category Test, (c) Critical Flicker Fusion Test, (d) Tactual Performance Test, (e) Rhythm Test,

(f) Speech-sounds Perception Test, (g) Finger Oscillation Test, (h) Time-sense Test, (i) Trail Making Test, (j) Aphasia Screening Test, and (k) Minnesota Multiphasic Personality Inventory. Each test in the battery is independent and may be administered separately from each other. However, it is generally advised to administer a certain number of tests in order to derive an impairment index. On the basis of recent reviews, the Critical Flicker Fusion Test and the Time-sense Test have been dropped from the Halstead-Reitan Battery, because of their diagnostic inefficienty (Ball, 1981).

Scoring and interpretaton of the Halstead-Reitan Battery is flexible. Scoring varies with the test, time to complete the test, errors, and number of correct responses. An impairment index with values ranging from 0 to 10 (least to most) was devised by Halstead for making gross diagnosis. Normal brain functioning is assumed when the Impairment Index is at 5 or lower. Clinical interpretation of the test scores is guided by level of performance, pattern of performance, specific behavioral deficits, and comparison of right and left brain functioning (Goldstein, 1984).

Critical Evaluation of the Halstead-Reitan Battery. Although the Halstead-Reitan Battery claims to be a sound diagnostic instrument, there are several practical limitations. First of all, the battery is too long, requiring approximataely six to eight hours of administration time. Second, the tests do not have any theoretical foundation. They are based primarily upon empirical (clinical) work. Therefore, the battery does not generate new ideas regarding brain-behavior relationships (Goldstein, 1984). Third, the Halstead-Reitan tests are not very specific about the assessed cerebral function

(Luria, 1973). Fourth, the presence or absence of brain damage is determined by an Impairment Index which has been found to be less accurate than the informaton obtained from clinical judgment, interview, and medical history (Goldstein, 1984). Fifth, the Battery is not very comprehensive because it neglects the area of memory completely. Sixth, the Halstead-Reitan tests are not suitable for thorough examination of patients with various sensory or motor handicaps (Butters, 1983). Seventh, the test battery is not sensitive enough to distinguish between brain damage and schizophrenia (Heaton & Crowly, 1981). Finally, the tests used in the battery are highly correlated, therefore the information obtained from the various tests is generally redundant.

The Luria-Nebraska Neuropsychological Battery (LNNB)

This is a recently developed comprehensive neuropsychological assessment battery mainly derived from the works of Luria, a Russian neuropsychologist who had developed highly sophisticated clinical methods for evaluating organic patients. Luria's work became better known in the west with the publication of his books in the English language (Luria, 1966, 1970, 1973). Before describing the LNNB, it may be appropriate to discuss Luria's theoretical model.

Luria's Model of Neuropsychological Functioning. Luria (1973) believes that the sensory and motor functions of the human brain are sufficiently localized. Complex functions, on the other hand, are under coordinated control of a number of brain structures with highly specific functions. In contrast to the traditional neuropsychological approach focusing upon brain localization, Luria's theoretical model emphasizes the functional organization of the brain. This functional

system attempts to describe a given behavior in terms of the hypothesized chain of interlinked parts of the brain. As explained by Golden (1981):

> "...each brain area involved in a behavior forms a link in the overall chain. All behaviors are presented by functional systems with multiple links representing each of the three major units of the brain." (p.193)

Luria conceptualizes the human brain as consisting of three main blocks or units of basic functions. They are: (a) the Reticular System, (b) the Sensory Reception and Integration Unit, and (c) the Planning, Evaluation, and Motor Output Unit. The first unit is located in the upper and lower parts of the brain stem reticular formation. This structure controls wakefulness and memory traces, and regulates the energy level of the cortex. In addition to the general functions, the first block provides stability to the organization of various cortical processes.

The second unit is situated in the rear side of the cortex and has highly specific functions, such as analysis, coding and storage of information. In contrast to the first block, injury to the second unit produces specific functional deficit, for example, loss of vision or hearing. The cortical areas in the second block have a hierarchical organization consisting of a primary zone (records sensory input), a secondary zone (organizes and codes information), and a tertiary zone (combines overlapping data from different sources for organized behavior). The frontal lobes are included in the third unit which regulate attention and concentration, and are involved in complex cognitive functions. Luria (1970) asserts that although simple sensory and motor functions are relatively uninfluenced by the frontal lobes, they play major roles in complex behavioral processes.

Luria's theory of functional systems suggests that the brain is neither completely localized, nor completely holistic. Different parts of the brain have specialized functions, yet there exists a dynamic system of interrelationships between brain structures. Thus, the three blocks or units are involved in all functional systems. Injury to a particular brain area may not result in a specified behavioral dysfunction. It is important to note that although Luria's model is highly sophisticated, he devised clinical procedures to break the complex functions into simple testable components so that behavioral deficits could be linked to specified brain injury.

Test Development. The innovative approach presented in Luria's work, attracted Anne-Lise Christensen to study with Luria in the Soviet Union. She was the first person to write a book explaining Luria's techniques. She also published a test kit containing a manual of instructions and test cards on the basis of theory and materials used by Luria and his coworkers (Christensen, 1975). However, Christensen's kit did not provide any information about important psychometric properties, such as standardization criteria, norms, reliability, and validity.

In the late 1970's, Golden and his associates undertook the task of developing a psychometrically respectable instrument in respect of standardization, reliability and validity incorporating Luria's methods (Golden, Hammeke, & Purisch, 1978; Purisch, Golden & Hammeke, 1978). Golden's pioneering work stimulated the interest of many other serious investigators in the field of neuropsychology and soon a massive literature became available. In 1980, Golden and coworkers published the Luria-Nebraska Neuropsychological Battery

(LNNB) with test materials and an excellent manual. The popularity of this test can be assessed by the fact that within five to seven years the Luria-Nebraska has become a leading competitor of the Halstead-Reitan Battery. Since its publication, the battery has been used extensively in clinical and research studies.

The Luria-Nebraska differs from the Halstead-Reitan in several ways. The administration time is considerably less; it takes about two or two and one-half hours only to administer the entire LNNB. It is well-standardized in test items, administration, and scoring. It covers broad as well as specific areas of neuropsychological deficits. Furthermore, precise location of brain damage is claimed to be possible. The distinctive characteristic of this battery is the use of individual items instead of the test as a unit (Golden, 1981).

Cross-validation studies reported by Golden and his associates are generally favorable (Golden et al., 1979, 1980). Content and discriminant validity studies are also satisfactory. The test was able to differentiate a miscellaneous group of brain damaged patients from normal controls and schizophrenics (Golden, Hammecke, & Purisch, 1978). Predictive validity of the test has not yet been established (Goldstein, 1984). Studies on construct validity are not sufficient yet to claim LNNB's adequacy (Goldstein, 1984; Lezak, 1983).

The principal advantage of the Luria-Nebraska is that it is based on the theoretical principles of neuropsychological functioning which makes interpretation logical. It is also flexible, easy to administer and inexpensive (Anastasi, 1984; Goldstein, 1984; Lezak, 1983).

Critical Evaluation. Since its origin in 1980, the LNNB has

been subjected to considerable debate among researchers. Both positive and negative arguments have been presented. The criticisms of the LNNB are both specific and general. There are two general criticisms. First, the Luria-Nebraska Battery does not reflect Luria's thinking accurately, and as such his name should not be used (Goldstein, 1982). The second criticism is concerned with methodological issues. Adams (1980) criticized subject selection, test development and qualification methods with the suggestion that further developmental studies were needed before making clinical applications of the battery. Spiers (1981, 1984) is critical of the LNNB's claim of providing comprehensive neuropsychological assessment and discriminating between various neurological populations. The battery also has been considered diagnostically unreliable (Adams & Brown, 1980; Crosson & Warren, 1982; Delli & Kaplan, 1982). Crosson and Warren (1982) found that LNNB does not provide sufficient data to identify aphasic disorders and misidentifies the side of the lesion in aphasic patients. Another specific criticism is that the battery is heavily language-oriented. The memory scale is also criticized because comprehensive assessment of memory is not possible with LNNB (Russell, 1981). Golden has attempted to improve the battery by adding several items involving delayed recall in the next version of the battery (Goldstein, 1984).

Other critical issues mentioned in recent literature are concerned with the proper utilization of LNNB in clinical settings. However, Goldstein (1984) believes that LNNB "...may be so used as long as inference made from it do not go beyond what can be based on the available research literature" (p.206). Stambrook (1983) is

correct when he says: "The clinical utility of the LNNB does not depend upon either the publisher's and test developer's claims, or on conceptual and methodological critiques, but upon carefully planned and well-executed research" (p.266).

Notwithstanding the fact some of the criticisms of LNNB are very strong, it is too early to make a definitive evaluation of this battery in the absence of controlled studies on normals as well as on organic patients (Goldstein, 1984; Stambrook, 1983).

As discussed above, both the batteries have advantages and disadvantages. The Halstead-Reitan is well established and detailed, but it is quite lengthy, cumbersome, and neglects some specific areas, specially memory. On the other hand the Luria-Nebraska is brief, but fairly comprehensive. However, at present LNNB is subject to considerable controvery among researchers. The claim that this battery is based on Luria's theory and methods has not beeh verified (Goldstein, 1984).

Summary of the Literature Review

Normal Aging Process

Since age associated changes in appearance, physical and mental capacity, and behavior do not seem to follow any typical pattern, researchers in the field suggest conceptualization of aging at three levels: (a) biological aging, (b) psychological aging, and (c) sociological aging. It is assumed that the process of aging involves a long and gradual decline in all the three areas. Individual differences in aging may be explained as due to the complex

interaction of genetic endowment, psychological factors and social conditions.

Most gerontological studies have focused upon age differences in cognitive functioning. Some researchers observe cognitive decline with advanting age, while others believe that cognitive abilities are preserved well into the 80s and 90s. It is apparent that considerable research is needed to achieve proper understanding of the normal aging process.

Although there is little agreement about the exact nature and amount of cognitive, affective and behavioral changes with advancing age, most studies suggest significant relationships between age and performance on neuropsychological tests. This relationship is generally acknowledged to be the function of the normal aging process. The decline may start earlier in life, or may remain confined to only selected areas of neuropsychological functioning. However, some gerontological research suggests that test performance of those over 65 frequently falls in the range of brain dysfunction; although education and intelligence clearly confound this interpretation.

Abnormal Aging Processes

Alzheimer's Disease (AD), Multi-infarct Dementia (MID), and Depression (DEP) are common disorders in the elderly population. Impairment in cognitive functioning, such as intelligence, memory, abstract thinking, and judgment are observed in all the three disorders.

Alzheimer's Disease (AD) is a serious disorder of the later decades of life producing diffuse deterioration of thought, memory,

and other major cognitive functions. Although early symptoms of AD are typically subtle, several neuropsychological changes associated with frontal, temporal and parietal areas of the brain become conspicuous as the disease progresses. Behavioral symptoms of the frontal lobe damage consist of indifference to social decorum, loss of personal care, lack of interest in the environment, and poor insight. Tactile agnosia and aphasia are two major behavioral symptoms of the parietal lobe dysfunction. The temporal lobe damage may be responsible for speech and language difficulties resulting in simplification of language during the advanced stages of AD. Although the etiology of AD is not clearly established, the pathology of this syndrome includes the presence of senile plaques, the loss of neurons in the nucleus basilis and depletion of cholinergic activity.

Multi-infarct Dementia (MID), is characterized by spotted areas of dead brain tissue producing focal cognitive impairment and erratic swings in behavior. That is, the brain lesions in the Multi-infarct Dementia are patchy rather than uniform. Hence, the dementia is related to the brain areas where infarct has occurred. The etiology of Multi-infarct Dementia is considered to be the hardening and narrowing of the cerebral blood vessels, leading to chronic shortage of blood to the brain which, in turn, contributes to the mental deterioration.

The depressive disorder in old age is very similar to dementia, since both have symptoms of disorientation, loss of memory, and impaired judgment. However, research findings suggest that neuropsychological deficits in Depression (DEP) are not as pronounced and extensive as they are in AD and MID patients. Further, successful

treatment of depression is likely to restore normal neuropsychological functioning. This suggests that there is no permanent damage to any cortical area in depression.

It is apparent from the review of the literature that cognitive impairments in older adults range from the mild in normal aging to the more severe in degenerative brain diseases. In the case of depressive disorder, the clinical symptoms of cognitive decline are generally mild, but transient without any organic involvement. However, the research literature has failed to provide a clear-cut picture of the expected changes in various facets of cognitive functioning in both normal and abnormal aging. The present state of our scientific knowledge is not very helpful in the differential diagnosis of the dementing process, nor does it present a coherent theoretical explanation of cognitive deficits associated with aging, organicity, and psychopathology.

Perhaps the main reason for the lack of precise knowledge about the nature and extent of cognitive deficits is the limited scope of the existing studies in this field. In this connection, it may be noted that most researchers have selected primarily memory and intelligence to study cognitive changes associated with normal aging and dementing processes. Another gap in the existing gerontological research is related to the selection of psychometric instruments for the purpose of making comprehensive and objective assessment of higher nervous system dysfunction. As discussed before, none of the studies has employed a comprehansive neuropsychological test battery so that our knowledge of cognitive impairment is limited to only a few functions, such as memory and intelligence.

The present research, therefore, is an attempt to fill the gaps discussed above in understanding the brain-behavior relationship in normal and abnormal aging. Specifically, by using a comprehensive neuropsychological battery, such as the LNNB, it would be possible to determine the locus of higher nervous system dysfunction indicating the lobe(s) and hemisphere(s) affected in the selected subject groups. The presence of diffuse or focal impairment can also be assessed more acturately through a standardized comprehensive neuropsychological battery. Since confidence in the data on any research issue depends to a large extent upon the measuring insturments, the present research uses four additional commonly used neuropsychological tests, namely Halstead-Wepman Aphasia Screening Test, Trail Making Test, Wechsler Memory Scale, and Wisconsin Card Sorting Test, measuring specific cognitive functions that are also included in the LNNB. This is likely to provide more reliable information about the nature of cognitive deficits in normal aging, depression, and dementia.

Hypothesis and Expectations

As discussed in the review of the literature, there is considerable deterioration in the neuropsychological functioning of elderly Alzheimer's, Multi-infarct Dementia, and depressive patients compared to healthy old persons. However, the precise nature and degree of impairment is not clearly known. In the early stage, degenerative brain diseases produce symptoms similar to those observed in normal aging. The situation becomes much more confusing in later stages, because of overlapping symptoms of different dementias and

psychopathological conditions (Lauter, 1985; Reisberg, 1983). For example, while most studies have shown decline of general intellectual functioning in dementia (e.g., Lorning, 1985; Masur & Fuld, 1985), some investigators could not find any differences between early stages of Alzheimer's dementia and normal aging (e.g., Botwinick, 1951; Whitehead, 1973). Similarly, the precise nature of memory loss in degenerative dementia, depression, and normal aging is not well-established (e.g., Weigartner et al., 1981; Wilson et al., 1983). Although intelligence and memory have been studied extensively, there are very few research studies on the impairment of sensori-motor, language and communication functions in the elderly population. This research, therefore, planned to assess a wide variety of cognitive functions in degenerative dementia and normal aging with the help of a comprehensive neuropsychological instrument, namely the Luria-Nebraska Neuropsychological Battery (LNNB), and four other neuropsychological tests. Consistent with the general direction of the findings reported in the literature and rationale discussed above, the following hypothesis was tested in the present research:

Compared to the normal healthy elderly, there is significant neuropsychological impairment in the elderly suffering from degenerative brain disease (Alzheimer's and Multi-infarct Dementia) and depressive disorder.

The literature relating to the neuropsychological bases of Alzheimer's Disease, Multi-infarct Dementia, and Depression suggests that different areas of the brain are affected adversely in respect of

lesion sites and severity of impairment (Kuhl et al., 1985). Thus, in Alzheimer's Disease, neuronal degeneration in the cortex is global and most severe. In Multi-infarct Dementia, on the otherhand, brain dysfunction is focal and severe, thereby producing dementias only in those areas of the brain where infarcts have occurred. In the case of Depression, although the impairment is global, it is much less severe compared to AD and MID patients. Yet the neuropsychological deficits in Depression are generally more severe than in normal aging.

The gerontological literature reports inconsistent findings about the relationship between age and performance on neuropsychological tests. Some studies suggest progressive decline in cognitive functions with advancing age (e.g., Bak & Green, 1980; Benton, Eslinger, & Damaso, 1981), while others report intact neuropsychological functioning even in the 80s and 90s (Thompson & Marsh, 1973).

The specific findings with respect to the Alzheimer's Disease, Multi-infarct Dementia, Depression, and normal aging prompted the following expectations in this research:

1. The Alzheimer's patients will show marked global neuropsychological impairment compared to the Multi-infarct patients who will show patchy or focal impairment.

2. The depressed elderly will perform significantly better on all neuropsychological tests compared to the Alzheimer's and Multi-infarct patients.

3. There will be no significant difference in neuropsychological functioning between normal healthy young elderly (55 to 74 years) and normal healthy old elderly (75 years and over).

CHAPTER III

METHOD

Chapter III contains an overview of the subject selection criteria and logical decisions which were made in this regard. Similarly a comprehensive overview of the specific instruments is offered. Finally the procedure for data collection is provided.

Subjects

Participants in this research were 65 elderly persons consisting of nineteen Alzheimer patients (AD), eleven Multi-infarct patients (MID), fourteen depressives (DEP) and twenty-one healthy normals. However the data from twelve subjects were discarded because the responses were unscorable. These subjects were unable to complete the entire test battery due to one or more of the following reasons: loss of interest, lack of comprehension of the test items, and discharge from the hospital before the testing was completed. Out of twelve discarded subjects, seven were from the Alzheimer's group, three from the Multi-infarct and two from the depressive group. Thus, a total of 53 subjects (twelve AD, eight MID, twelve DEP and twenty-one normals) provided complete data for comparison purposes.

Initially, it was planned to divide each of the four groups into two subgroups based on chronological age--(a) 55-74 and (b) 75 and over. The rationale for dividing the groups on the basis of age was mainly to see whether age has differential effects on the test performance of the four groups. Since some studies suggest that

normal young elderly have less cognitive decline than normal old elderly, it is reasonable to assume that the young-old demented persons have less impairment compared to the old-old patients, even when both groups are at the same stage of the disorder. However, except for the normal elderly, it was not possible to obtain even three suitable subjects in each of the two age ranges for all the three groups. The normal elderly subjects were divided into two groups: young old (55-74) and old old (75+). This grouping was suggested by Neugarten (1975), and has been recognized as a useful age distinction by researchers in the field. There were eleven normal elderly subjects in the 55-74 age range and ten in the 75 and over groups. All subjects selected for this study were right handed male and female white Caucasian.

The subjects ranged in age between 55 to 93 with a mean age of 72.77 and SD of 9.09. Tables 3 and 4 show descriptive data regarding age, education, sex, marital status, and occupation of the subjects. There were no significant differences between the groups with respect to the above-mentioned demographic characteristics. It may also be noted that the distribution of male and female subjects in this study is consistent with the general finding that Alzheimer's Disease is more prevalent among women, whereas more males suffer from Multi-infarct Dementia (Kay, 1964; Reisberg, 1983).

The normal elderly were volunteers living independently in the community. They were approached through The Society of Retired and Semi-Retired in Edmonton. Only those volunteers whose medical history did not reveal any physical or mental health problems, such as myocardial infarction, diabetes mellitus, peripheral vascular disease,

Table 3

Means and Standard Deviation of Age and Education for the Five Groups

Age	N	Range	Mean	SD
Normal I 55-74	11	57-73	67	9.82
Normal II 75+	10	77-91	81	4.47
DEP	12	56-93	70	9.54
AD	12	55-87	70	9.82
MID	8	59-88	75	9.49
Education				
Normal I 55-74	11	9-18 years	13 years	2.40
Normal II 75+	10	8-18 years	12 years	3.13
DEP	12	7-16 years	11 years	3.17
AD	12	6-13 years	10 years	2.10
MID	8	7-18 years	11 years	3.54

Table 4

Number and Percentage of Sex, Marital Status and Occupation for the Five Groups

	Normal I		Normal II		DEP		AD		MID	
	No.	%	No.	%	No.	%	No.	%	No.	%
SEX										
Female	9	82	4	40	10	83	8	67	2	25
Male	2	18	6	60	2	17	4	33	6	75
MARITAL STATUS										
Married	4	36	2	20	5	42	6	60	5	62
Widow	4	36	8	80	7	58	4	40	3	38
Unmarried	2	18	0	0	0	0	0	0	0	0
Divorced	1	10	0	0	0	0	0	0	0	0
OCCUPATION										
Housewives	1	9	4	40	3	25	2	17	1	12
Skilled Worker	1	9	2	20	2	17	4	33	2	25
Semi-Prof.	5	46	0	0	0	0	3	25	2	25
Professional	4	36	4	40	7	58	3	25	3	38

head trauma, syncopal episodes, progressive memory impairment, or psychiatric disorders served as subjects. Also, they did not have any history of alcohol or drug abuse.

The subjects belonging to the Alzheimer's, Multi-infarct, and depressive groups were selected during a period of ten months from the in-patient population of three general hospitals in Edmonton: Edmonton General Hospital, Misericordia Hospital, and Alberta Hospital. However, three Alzheimer's and two Multi-infarct patients living at home volunteered to participate in this research project through the Alzheimer's Society of Edmonton. Also, five depressive patients were obtained through out-patient referrals at Misericordia Hospital.

The diagnostic criteria used in this study for selecting appropriate patients in the three disease categories are detailed below.

Alzheimer's Disease (AD)

The Alzheimer's Disease patients met the criteria for Primary Degenerative Dementia described in DSM III (American Psychiatric Association, 1980). They all had undergone at least a two to four-year period of gradual progressive impairment of memory, abstract thought, and other intellectual capacities. But no one had reached a state of severe impairment. Also none of these patients had a history or current laboratory evidence of cerebrovascular accident, hypertension, myocardial infarction, diabetes mellitus, vascular disease, skull trauma, syncopal episodes, epilepsy or thyroid dyscrasia. No evidence of abnormal test results including Serological Screening for Syphilis, Serum Vitamin B12 levels and Thyroid functions was found.

Also none of these patients had any history of psychiatric problems. No drug abuse or alcohol problems were reported in the patient's medical chart.

Findings of Computerized Axial Tomography (CT Scan) were either normal or showed signs of diffuse cortical atrophy. There was no evidence of focal or unilateral disturbance. Also, the findings of Electroencephalogram (EEG) were either normal or showed diffuse anomalies throughout the cortex. The general physical state of health as recorded was normal.

Multi-infarct Dementia (MID)

All subjects with Multi-infarct Dementia met the general criteria of DSM III (American Psychiatric Association, 1980) for Degenerative Dementia and specific criteria for Multi-infarct Dementia. The diagnosis was based on psychiatric, medical and comprehensive laboratory screening tests. All patients showed signs and symptoms of stepwise deterioration of cognitive functioning with patchy distribution of deficits. The medical and neurological examinations clearly showed evidence of focal neurological signs and symptoms of this disease. Also all subjects had a history and medical evidence of cerebrovascular accident. Results of Computerized Axial Tomography (CT Scan) showed mild to moderate cortical atrophy. Evidence for EEG and Skull X-ray supported the diagnosis of MID. The above-mentioned clinical symptoms were observed to be present for two to four years in each case. None of the patients had alcohol, drug and psychiatric problems.

Depression (DEP)

The selection of the depressive patients was made on the basis

of the diagnostic criteria specified in DSM III (1980, p.213) for Major Depression. In addition, clinical observations and impressions of the attending psychiatrists were also considered in selecting the patients. The DSM III criteria for depression are the following.

A. Dysphoric mood or loss of interest or pleasure in almost all usual activities and pastimes.

B. At least four of the following symptoms have each been present nearly every day for a period of at least two weeks.

1. poor appetite or significant weight loss
2. insomnia or hypersomnia
3. psychomotor agitation or retardation
4. loss of interest and pleasure in usual activities
5. loss of energy
6. feelings of worthlessness, self-reproach or excessive or inappropriate guilt
7. complaints or evidence of diminished ability to think or concentrate
8. suicidal ideation or suicidal attempts.

As suggested in DSM III, the patients' records were checked to exclude those who had mood-incongruence, delusion or hallucination, schizophrenia, schizophreniform disorder, paranoid disorder, organic mental disorder. In addition, the patients did not have any drug or alcohol problems.

Selection of the patients in this category was also based upon medical and laboratory tests to rule out depression and dementia due to any physical and neurological disease such as Alzheimer's, Multi-infarct, Parkinson's, Picks Intracranial Tumors, Hydrocephalus, etc. The medical tests also included CT Scan, EEG and skull X-ray for all patients. At the time of the testing, no depressive patient was

receiving electroconvulsive treatment (ECT). Although inevitably all patients were on some sort of medication, the attending physician established that none of the patients selected for this research were on any medication that might affect their test performance.

All patients (AD, MID, DEP) were diagnosed with the disease for at least two years, and were classified in the early or middle stage of the disease. No patient with the advanced stage of the disease was included. The relevant information regarding diagnosis was obtained from medical records of each patient. As already indicated, the diagnostic decisions were made in collaboration with the physicians and psychiatrists in-charge of the patients on the basis of the patients' medical record showing detailed history of the disease, neuroradiological examinations and histopathological data. Only those patients were selected for this study who met the clinical criteria for AD, MID, and Depression without any doubt or ambiguity. This procedure of subject selection is consistent with the studies reported in the literature (Erkinjuntti et al., 1986; La Rue et al., 1986; Perez et al., 1975, 1976).

In what follows a brief statement will be made on the decisions taken firstly, not to include detailed data concerning neuroradiological findings, secondly, not to use psychological tests for the measurement of depression, and thirdly, not to document the medication history of each patient.

Neuroradiological findings

The actual neuroradiological data are not reported in this research because of the following reasons:

1. At present there are no definite medical, neurological and neuropsychological markers that can accurately identify different forms of dementia (Davis, 1984; Gottfried, 1985). For example, although cortical atrophy shown on CT scans correlate to some extent with the degree of behavioral impairment observed in clinical diagnosis of AD (de Leon et al., 1980; Fox et al., 1978; Roberts et al., 1976), they are also found to be age related (Earnest et al., 1979; Hughes & Gado, 1981). Wilson et al. (1982) also found that CT scan failed to differentiate between Alzheimer's patients with normal healthy age-matched individuals. Measures of sulci widening and ventricular enlargement obtained through CT scans are usually considered insufficient for making the diagnosis of Alzheimer's Disease (Jacoby, Levy & Davison, 1980; Task Force Report, 1980). In some AD patients, no abnormalities on CT scans are detected (Naeser et al., 1980), while in others diffuse atrophy may be present without the corresponding degree of cognitive deficit (Gigler & Steinman, 1981; Koszaniak, 1986).

The diagnosis of MID using CT scans also has severe limitations (Koszaniak, 1986). Roberts et al. (1978) reported that CT scanning was only able to identify 20% of the MID cases. Although CT scan measures may be helpful in identifying small areas of infarctions in the brain, they have not been very helpful in ruling out mass lesions (Wilson et al., 1982). It is also stated by La Rue, Dessonville and Jarvik (1985), that there is wide recognition that lesions smaller than 1 millimeter in size can not be detected on the CT scans. This points out also, that small infarctions (less than 1 mm) are likely to have behavioral implications.

There are very few cases of MID patients with striking signs of focal brain damage. The striking focal signs are present in relatively mild impairments (Fuld, 1983). Many researchers report that in MID, the dementia results from an accumulation of bilateral cerebral hemispheric infarctions due to recurrent minor strokes. Perry and Perry (1982) for example state:

> The majority of cases of Multi-infarct dementia in the elderly prove to have several cerebral infarcts of a moderate or large size rather than multiple separate lesions, and in most cases both hemispheres are affected -- although not necessarily equally or symmetrically (p. 37).

Similar observations have been made by Meyer (1983), and Meyer, Miyakwa, Ishihara et al. (1977). Thus, most researchers do not find CT scans sensitive enough to identify cerebral hemisphere infarcts (e.g., Bondereff et al., 1981; Naeser, 1980). Differentiating Alzheimer's Disease from vascular dementia is difficult on the basis of CT scan, since CT scan often shows central atrophy in both diseases (Gustafson, 1985). Its diagnostic utility in differentiating AD or MID patients from normal and depressed older persons is still questionable (Fox et al., 1979; Koszaniak, 1986).

In a critical review of the pathological studies on AD and MID covering the past four decades, Liston and La Rue (1983) report that at present there are no clear diagnostic criteria, especially for MID. In view of the limitations of currently available neurophysiological techniques, most researchers suggest the use of clinical diagnosis of dementia, particularly in AD and MID. In a recent comprehensive review of the literature of this topic, La Rue, Dessonvelle, and Jarvik (1985) assert:

> Because of the limitations of current neuroradiologic and electrophysiologic techniques, the diagnosis of dementia is made on clinical grounds, and once specific etiologies have been ruled out, a premium is placed on an accurate description of the extent and pattern of behavioral impairment (p. 678).

The qualitative rating scale, namely the Hachinski Ischemic Index (Hachinski et al., 1975) was also not used because it is composed of ambiguous items similar to those in DSM III. Also, the accuracy of this measure is yet to be established firmly (Loeb, 1980). In a recent study Wade et al. (1987) assessed the reliability of clinical diagnosis of dementia of the Alzheimer type against the criterion of autopsy findings. They reported 87% accuracy for the clinical diagnosis in their logitudinal study. They also reported that the Ischemic Scale could not discriminate well between Multi-infarct dementia and dementia of the Alzheimer type.

2. Since the purpose of this research was not to correlate the neuropsychological test results with the amount and nature of neurophysiological changes, no attempt was made to report these measures. The main purpose of this study was to compare the nature and amount of cognitive impairment in five distinct groups. It was, therefore, not considered necessary to correlate the psychological test performance with physiological measures. Almost all psychological studies on dementia and depression have ignored neurophysiological data in studying the nature of cognitive functioning (e.g., Bornstein, 1986; Bucht & Adolfsson, 1986; Erkinjuntti, Laaksonen, Sulkava, Syralainen & Palo, 1986; Moses & Pickett, 1986; La Rue et al., 1986; Lorning & Largen, 1985; Perez et al., 1975; Perez et al., 1976; Storrie & Doerr, 1984; Vitaliano, 1986; Lewinsohn, 1986).

Measurement of Depression

As stated earlier, the depressed subjects were selected on the basis of the DSM III criteria for Major Depression. The reason for this decision was twofold: on the one hand there is the question of appropriateness of self-rating scales, on the other hand the lack of norms for both self-rating and other scales for the elderly population.

Self-rating scales such as the MMPI and the Beck Depression Inventory require generally intact cognitive functions; moreover, these scales have so many deficiencies, even for younger populations, and in addition they lack norms for the elderly populations. For all these reasons they are not recommended for the measurement in the elderly (Blazer, 1982; Mayer, 1978). More specifically, several studies have shown that it is difficult to make normal vs depressed distinction among the elderly population when a self-rating scale is used (La Rue et al., 1985). It has been found that even the normal elderly are likely to be mislabelled as depressed on self-rating scales. Researchers have also reported that older persons perform differently than young middle-aged adults on virtually all psychodiagnostic instruments presently available (Albert, 1981; Zarit et al., 1981).

In the absence of sufficiently normed, reliable and valid psychological tests for the measurement of depression in the elderly, the DSM III criteria were considered more appropriate for selecting patients in this category.

Medication

No attempt was made to document the drug history of each patient, since the patients were selected in collaboration with the attending physicians who were certain that the dementia shown by these patients existed independently of any drug effect. No subject in the dementia and depression groups was selected if there was any question about possible drug confound. Most published psychological and neuropsychological studies on dementia and depression do not provide detailed drug history of the patients, particularly when it was used as the exclusion criteria (e.g., Bornstein, 1986; Erkinjuntti et al., 1986; La Rue et al., 1986; Lorning & Largen, 1985; Perez et al., 1975, 1976; Martin, Brouwers, Lalonde, Cox, Teleska, Fedio, Foster & Chase, 1986).

Thus, it should be noted that all patients who participated in this study had received a definite diagnosis on the basis of various neurodiagnostic criteria presently used in the teaching hospitals of the University of Alberta (please see pages 86-88 for the details of the criteria). Since the purpose of this research was not to see the effect of medication on cognitive functioning, detailed drug history of each patient was not considered relevant. The only consideration was that no patient in any group had the clinical syndrome of dementia and depression associated with medication.

Although electroencephalogram (EEG) has been used in the diagnosis of dementia, most researchers caution in interpreting the results due to the limitations of this procedure (Hutton, 1980). The EEG has been found to have little diagnostic value for assessing MID

patients. It has been reported that brain infarcts may not produce abnormal EEG specially when infarcts are small and farther from the cortical surface (Kaszaniak, 1986).

Instruments

The psychological tests used in this research are briefly described below.

1. The Luria Nebraska Neuropsychological Battery (LNNB)

This test is based on the methods and techniques developed and utilized by the famous Russian neuropsychologist, A.R. Luria, in his clincal work with patients. Luria's techniques that were collected and organized by Christensen (1975) have been converted into 269 test items in the Luria-Nebraska (Golden et al., 1980). These items have been grouped into eleven content scales measuring motor functions, rhythmic and pitch abilities, tactile and visual functions, receptive and expressive speech, reading, writing and arithmetic skills, memory and intellectual processes. In addition to these eleven content scales, the LNNB also provides three other scales; Pathognomic, Left Hemisphere and Right Hemisphere. The Pathognomic scale measuring the subject's overall impairment has been found to be highly sensitive to brain dysfunction. The Left and Right Hemispehre scales measure the basic tactile and motor functions for the two sides of the body. These two additional scales are useful in discriminating laterality of brain dysfunction.

The present form of the Luria-Nebraska Battery contains 269 items. Each item represents specific and relevant skills, and the items differ from each other in mode of stimulus input, response

mode, and difficulty level. The items are scored on the basis of accuracy, speed and quality of the response. The scores range from 0 to 2 where a score of 0 indicates normal performance, 1 borderline, and 2 clearly abnormal performance. The obtained raw scores are converted into T scores with a mean of 50 and standard deviation of 10. The test manual provides clear and extensive information regarding administration, scoring and interpretation (Golden et al., 1980).

The Luria-Nebraska scoring procedure involves the use of critical levels. These are baselines determined by a formula that takes into consideration age and educational level of the testee. According to the manual, a normal person's T score greater than 60 is indicative of brain damage, whereas in psychiatric patients a T score greater than 70 is suggestive of brain damage.

Golden and his associates (1981) found that 252 out of 269 items on the LNNB are able to discriminate between brain damaged and normal persons at the .05 level of confidence. The remaining 17 items are significant at .20 level.

Golden and his associates have reported impressive reliability data to support LNNB's psychometric superiority over other tests measuring similar functions. In general, the reliability coefficients obtained through split-half and test-retest techniques range from .77 to .96 for different scales. Reading and Arithmetic scales have reliability coefficients of .95 and .96 (Golden et al., 1978) respectively, whereas the lowest reliability coefficients have been reported for Tactile (.78) and right Hemisphere (.77) scales (Golden et al., 1980). Hammeke (1978), and Golden, Hammeke, & Purisch (1978,

1980) reported correlations ranging from .97 to .99 between two examiners for all the items on the battery. The reliability coefficients of individual scales are discussed in the test manual (Golden et al., 1980).

Diagnostic Validity of the LNNB

Brain-damaged vs Normal control. In their initial study Golden, Hammeke, and Purisch (1978) demonstrated that the LNNB was able to discriminate neurological from control subjects. They compared the performance of 50 hospitalized, neurological patients to the performance of 50 hospitalized patients with no neurological disorders. Sequential t tests revealed that the neurological group was significantly ($p < .05$) more impaired than the control group on 89% of the test items. A discriminant analysis using 60 items was 100% in classifying both groups. It was found that the accuracy of the individual scales to classify subjects correctly ranged from 74% (Expressive Speech) to 96% (Memory) in the control group (M=85%), and from 66% (Rhythm) to 86% (Expressive Speech) in the neurological group (M=74%). The combined hit rate for both groups ranged from 74% (Rhythm) to 86% (Pathognomonic Scale). A discriminant function analysis of 14 summary scales of the LNNB as a dependent measure was able to correctly classify 86% of the neurological group and 100% of the control group, with an overall hit rate of 93%.

Similar hit rates with respect to the LNNB's ability to discriminate neurological from control patients have been reported in a cross-validation study by Moses and Golden (1979). Another cross-validation study by Golden, Moses, Graber, and Berg (1981) using

additional 60 normal and 60 neurological patients resulted in a hit rate of 85% for the neurological patients and 83% for controls with a combined hit rate of 84%. Molloy and Webster (1981) reported that the application of the "critical level" procedure provided correct classification of 75% of the pseudoneurological group and 83% of the brain damaged group.

Brain-damaged vs. Schizophrenic. Purisch, Golden, and Hammeke (1978) have examined the effectiveness of the LNNB in discriminating between 50 hospitalized chronic schizophrenic and 50 hospitalized neurological patients. Two tailed t tests revealed that neurological patients were significantly more impaired on 72 of the 282 items. A stepwise discriminant analysis demonstrated 100% accuracy in classifying brain injured group with 40 items. The discriminant analysis on the 14 scales achieved 92% hit rate for schizophrenic group and 84% hit rate for neurological group yielding an overall hit rate of 88%. Moses and Golden (1980) did the cross-validation study on the results of Purisch et al. (1978) study, using the same neurological group as Moses and Golden (1979) with additional 50 schizophrenic patients of similar diagnosis. The discriminant functional analysis was able to yield 88% classification accuracy for the schizophrenic group and 86% classification accuracy for the neurological group. The overall hit rate was 87%.

Lateralization and Localization of Brain Damage. Osman et al. (1979) in their study investigated the ability of the LNNB to discriminate among lateralized and diffuse brain damaged-patients. Twenty patients were assigned in each group depending on the locus of brain damage (left, right, diffuse). A discriminant analysis based on

14 summary scales reported that the LNNB was able to accurately classify 59 of 60 cases (98% hit rate).

McKay and Golden (1979a, 1979b) developed scales for the left hemisphere, right hemisphere and 8 localization scales: (a) Right frontal, (b) Right sensorimotor, (c) Right parietal-occipital, (d) Right temporal, (e) Left frontal, (f) Left sensorimotor, (g) Left parietal-occipital, and (h) Left temporal. In a cross-validation study of 87 patients who had localized brain lesions in one of the eight area, Golden, Moses, Fishburne, et al. (1981), reported a hit rate of 92% for lateralization and 74% for localization.

An independent cross-validation study of the Luria Nebraska Neuropsychological Battery was conducted by Sears, Hirt, and Hall (1984). Their comparison groups consisted of brain-damaged individuals with evidence of (a) unilateral left hemisphere, (b) unilateral right hemisphere, or (c) bilateral diffuse brain lesions, as well as (d) normal control subjects. The results reveal that the LNNB was effective in discriminating brain-damaged from normal control subjects. Goldstein and Shelly (1984) also compared the discriminative validities of the Wechsler Adult Intelligence Test, Halstead-Reitan Battery and Luria-Nebraska Battery for predicting presence or absence of brain damage in the neuropsychiatric patients. The discriminant analysis revealed that percentage of correct classification was lower for the WAIS but was essentially equal for Halstead-Reitan and Luria-Nebraska batteries.

The LNNB's discriminative ability was also found satisfactory with specific neurological disorders, such as multiple sclerosis (Golden, 1979), alcoholism (Chmielewski & Golden, 1980), Huntington's

disease (Moses, Golden, Berger, and Wisniewski, 1981).

The above studies suggest that the Luria-Nebraska has respectable validity on many established groups (Goldstein, 1984).

Normative data for the elderly on LNNB

Normative data for the elderly on the LNNB were obtained by Spitzform in 1983. He administered the test to 14 normal elderly subjects with a mean age of 71.4 years. These norms are a valuable guide for interpreting the test results. However, we need more subjects for developing adequate and appropriate LNNB norms for the elderly. Spitzform compares the elderly normals with controls (42 yrs.) and brain-injured persons (44.3 yrs.).

Several validity studies using brain-damaged, schizophrenic, normals, and elderly subjects report an overall hit rate of 93% in detecting neuropsychological impairment (Golden et al., 1978; Hammeke & Purisch, 1978; Purisch, Golden, & Hemmeke, 1978; Spitzform, 1982). Factor analytic studies on the LNNB have confirmed the fact that the scales are homogeneous so that different scales measure separate neuropsychological functions (Golden et al., 1981). Golden and his associates have conducted several cross-validation studies confirming the value of the LNNB in discriminating brain damage from normal controls and other neuropsychological conditions (Golden et al., 1978, 1981; Moses & Golden, 1980; Osman, Golden, Purisch, & Blume, 1979).

Description of LNNB Scales

In the following section, a brief description of the clinical scales of the LNNB is presented. This description is based on the manual (Golden et al., 1980) and two other publications dealing with

interpretation of the LNNB (Golden, Hammeke, Purisch, Berg, Moses, Newlin, Wilkening & Puente, 1982; Moses, Golden, Ariel, & Gustavson, 1983).

Motor Scale. This scale contains 51 items for assessing a wide variety of both simple and complex motor skills. Items on this scale assess the individual's ability to organize, control and carry out motor movements involving upper extremities and oral areas. This scale is sensitive to lesions in the posterior frontal lobe. However, lesions of the temporal, parietal, and anterior lobes (prefrontal and premotor) are likely to cause elevated scores on the scale.

Rhythm Scale. This scale contains items that evaluate rhythm and pitch abilities as well as the ability to perceive and identify a number of tones in a sequence. The Rhythm scale assesses the ability to produce rhythmic patterns on verbal command. This scale is highly sensitive to attention and concentration disorders. Deficits in this scale are due to right hemisphere problems involving the temporal lobe (for understanding) and frontal lobe (for expression).

Tactile Scale. The Tactile scale evaluates a variety of simple and complex tactile and kinesthetic abilities. All items on this scale require identification by touch only. The person is asked to distinguish between hard and soft touch and to identify letters and numbers written on the back of the hands. This scale is most sensitive to injuries in the anterior parietal lobe of either hemisphere.

Visual Scale. This scale contains only fourteen items and is designed to evaluate the subject's visual and spatial skills. Simple recognition of familiar objects to complex visual-spatial processing

are included as tasks. Poor performance on this scale is indicative of right hemisphere involvement.

Receptive Speech Scale. The scale consists of 32 items specially designed to evaluate the ability to understand simple to complex grammatically structured sentences presented auditorially. The Receptive Speech Scale is much more easily elevated by damage to the left hemisphere. This scale may also be elevated by damage to the right hemisphere, especially the anterior temporal frontal areas.

Expressive Scale. In general, this scale assesses the individual's ability to express himself or herself orally. There are 42 items in this scale ranging from simple to complex words and sentences. The subject's spontaneous ability to discuss a topic in response to a picture, a story and a theme as well as the ability to organize words into intelligible statements are also evaluated in this scale. In general, expressive speech scores are sensitive only to injuries in the left hemisphere. It usually involves the temporal frontal area, especially the posterior two-thirds of the frontal lobe. If the complex items show the most deficits, then damage is more likely to be in the prefrontal area.

Writing Scale. This scale assesses the individual's basic writing skills. This includes simple spelling, the ability to copy words from cards, memory and dictation. Spontaneous writing skills are also evaluated on this scale. In general, disorders of writing localize to the temporal-parietal-occipital area, especially in and around the angular gyrus of the left hemisphere.

Arithmetic Scale. There are 22 items on this scale which assess knowledge of numbers and number concepts and the ability to

perform simple calculations. This scale is sensitive to the individual's educational and anxiety levels, and the ability to attend and concentrate.

Memory Scale. This thirteen-item scale assesses a variety of aspects of memory. It is basically involved with short-term and immediate memory. The scale also measures visual and nonverbal memory with or without interference. Extreme high elevation on this scale is almost always associated with either left or bilateral dysfunction.

Intellectual Processes. This scale involves brief assessment of a wide range of intellectual processes, such as sequencing, problem solving and abstract reasoning. There are a number of items similar to the WAIS Comprehension, Picture Arrangement, Arithmetic, Similarities, and Vocabulary subtests. The Intellectual Processes scale is highly sensitive to disorders in both hemispheres, but is most sensitive to disorders in the left hemisphere. Injuries to either the parietal lobe or the frontal lobe will cause maximum dysfunction. Poor performance on this scale without the presence of any psychiatric disorder is generally associated with prefrontal lobe dysfunction.

Pathognomonic Scale. This scale consists of 34 items from all the eleven scales of the LNNB that have been found highly sensitive to the presence of brain damage. The amount of elevation on this scale indicates the presence of brain impairment.

Right Hemisphere and Left Hemisphere Scales. These scales measure the lateralization of cerebral dysfunction. All items on the Right Hemisphere scale consist of left hand sensory or motor performance, whereas all items on the Left Hemisphere scale reflect

the right hand motor and sensory performance.

2. Halstead-Wepman Aphasia Screening Test

This is a highly respected and widely-used test incorporated in many neuropsychological assessment batteries (Lezak, 1983). The primary purpose of this test is to assess aphasic disabilities and communication problems. Copying a square, Greek cross and triangle without lifting the pencil from the paper, naming each copied figure, spelling each name, and repeating statements are some of the items in the test.

3. Trail Making Test

This is a two-part test (Part A and Part B) of visuomotor tracking involving visual concept, visual sequencing, motor speed and attention functions highly sensitive to brain injury. The subject is required to connect consecutively numbered and lettered circles as fast as possible, without lifting the pencil. Slow performance is believed to indicate brain impairment. Better performance on Part A than Part B suggests left hemisphere lesion in the brain, since the latter has randomly placed numbered and lettered circles.

4. Wechsler Memory Scale

This test has enjoyed considerable popularity in most neuropsychological assessment batteries, and consists of seven subtests. They are: (a) Personal and Current Information, (b) Orientation (time and place), (c) Mental Control, (d) Logical Memory, (e) Digit Span, (f) Visual Reproduction, and (g) Associate Learning (verbal retention). Items on the first two scales resemble typical mental status examinations. Performance on the Wechsler Memory Scale appears to deteriorate with advancing age as well as with

bilateral, diffuse, and left hemisphere lesions (Prigatano, 1978).

5. Wisconsin Card Sorting Test

This test was originally developed to measure "abstraction" ability and "shift of set" among normals. In a neuropsychological test battery, the Wisconsin Card Sorting Test has shown sensitivity to brain lesions, particularly in the frontal lobes (Tayler & Draw, 1974; Robinson et al., 1980). The test contains 64 response cards and two sets of four stimulus cards in red, green, yellow and blue colors with four different figures (crosses, circles, squares and rectangles) and four different numbers (one, two, three and four). The subjects' responses are scored for conceptualization, perseveration, failure to maintain set, and efficient learning. According to Robinson et al. (1980), the Wisconsin Card Sorting Test is a clinically useful instrument for detecting and localizing frontal lobe lesion.

6. Demographic Questionnaire

This is a locally constructed general questionnaire for the purpose of eliciting information about age, sex, education, occupation, medical and psychiatric history including records of hospitalization and medication (Appendix A).

Procedure

The subjects were tested individually by this researcher who has considerable experience with neuropsychological and psychological testing and in dealing with elderly persons.

In order to obtain the desired subjects from the hospitals, a letter explaining the nature of this research and requesting the types of patients needed for neuropsychological testing was sent to all the

attending physicians and psychiatrists. Several follow-up phone calls to the physicians, and personal visits to the hospitals were also made to ensure the cooperation of the physicians and the clinical staff. A copy of the letter is attached in Appendix B.

Before testing, written consent was obtained from the patients themselves or from their legal guardians. A copy of the consent form is attached in Appendix C.

Each patient was interviewed for about one-half hour by the examiner before testing in order to get a general clinical impression of the patients, particularly in respect of their physical and mental capabilities.

Most demented and depressed subjects were tested in the hospital premises with a few tested in their own homes. The Society for the Retired and Semi-Retired provided the space for testing the normals. However, some participants preferred to be tested in their own homes.

Medical history, laboratory findings and demographic information about the patients were obtained from the hospital records. For the patients tested in their own homes, the medical history and demographic information were obtained by interviewing the patients and spouses. If any further information was needed, this researcher had the family's permission to consult with the attending physicians.

All hospitalized subjects were tested between two to four weeks after their initial admission to the hospital. This procedure was followed to eliminate any effect of readjustment to the hospital environment, and to allow sufficient time to complete necessary

medical and laboratory tests.

The total time to complete the testing varied from patient to patient. In general, the AD and MID groups took between six to eight hours each to complete the entire test battery. On the average the depressive patients took between four to five hours each. The normal elderly subjects took between two and one-half to three and one-half hours only. All subjects were told to discontinue the testing any time they felt fatigued, discouraged and frustrated. However, after an hour of testing all subjects were given a short break of fifteen minutes.

Most normal comparison subjects took only one break of fifteen minutes, and continued with the testing until it was completed in one session. However, dementia and depressive groups required multiple sessions. The number of sessions required in the clinical groups varied from patient to patient. On the average, clinical subjects took four (DEP) and twelve (AD and MID) sessions to complete the testing. Each session lasted about 45 to 60 minutes. One or two breaks were usually required by the AD and MID patients in one session. The Alzheimer and Multi-infarct patients were usually not testable after an hour of testing in a day. The second session usually started the next day.

The tests were administered in the following order: (a) The Luria-Nebraska Neuropsychological Battery, (b) Wechsler Memory Scale, (c) Trail Making Test, (d) Halstead-Wepman Aphasia Screening Test, (e) Wisconsin Card Sorting Test. All tests were administered, scored and interpreted according to the guidelines provided in the respective test manuals.

CHAPTER IV

RESULTS

Originally, it was planned to analyze the data with appropriate multivariate methods, i.e., multivariate analysis of variance (MANOVA) and discriminant function analysis to confirm or reject the main hypothesis and expectations. These techniques are currently utilized in neuropsychological research for examining group differences and interrelationships between tests when there are several predictors or independent variables (Parsons & Prigatano, 1978; Pedhazur, 1982; Fletcher, Rice, & Ray, 1978). In a recent article, Franzen and Golden (1984) discuss the requirements of multiple regression techniques including the methodology of discriminant function analysis in neuropsychological research. They suggest that when the strength of the relationship between the predictors and the criterion is unknown, "it would be best to include at least ten times the number of subjects as the number of predictors" (Franzen & Golden, 1984, p.87).

In light of the recommendations discussed above, the data of this study have not been analyzed with multivariate techniques. The number of independent variables far exceed the number of cases in each of the five groups. Consequently, the data have been analyzed with ANOVAs and Neuman-Keuls procedure.

Following the suggestions of Moses et al. (1983) in their most recent interpretive manual, additional analyses of the LNNB data have

been made by the pattern analysis method utilizing the clinical, localization and factor scales. Since the purpose of the present research was to examine group differences in the cognitive functioning of AD, MID, Depression and normal controls, the test performance of individual subjects was not analyzed. This approach is consistent with the published studies on the topic (Erkinijuntti et al., 1986; Filley, Kelly & Heaton, 1986; Loring & Largen, 1985; Lewinsohn et al., 1986; Martin, Brouwers, Lalonde, Cox, Teleska, Fedio, Foster, & Chase, 1986; Perez et al., 1975, 1976; Vitaliano et al., 1986).

-- Comparison of the Groups on the Neuropsychological Measures

The primary hypothesis of this study predicted significant neuropsychological impairment in the Alzheimer, Multi-infarct and depressive patients in comparison with normal controls. Further, there were three expectations pertaining to the differential performance of the demented, depressed and normal subjects (see the Hypothesis and Expectations section). The data, were analyzed separately for each of the neuropsychological tests used in the study.

Luria-Nebraska Neuropsychological Battery

The means and standard deviations for each of the fourteen Luria-Nebraska scales in the five subject groups are presented in Table 5. One-way analyses of variance indicated that the groups were significantly different on all the scales ($p < .00005$). Post hoc comparison of the means using the Newman-Keuls procedure (p.05) for

Table 5

Means and Standard Deviations for the Five Groups on the Luria-Nebraska Neuropsychological Battery

Tests	Groups				
Luria -Nebraska	NOR I	NOR II	DEP	AD	MID
Scales	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)
Motor	34.91 (3.15)	44.70 (8.81)	52.00 (7.57)	91.00 (17.26)	91.00 (17.26)
Rhythm	37.55 (7.24)	49.00 (9.37)	62.92 (12.12)	87.92 (9.88)	81.50 (7.11)
Tactile	41.82 (5.08)	48.40 (7.23)	51.33 (7.90)	85.18 (11.99)	84.50 (13.18)
Visual	41.27 (7.42)	44.80 (6.03)	55.33 (6.73)	77.42 (11.02)	76.00 (14.64)
Receptive Speech	33.54 (3.14)	37.40 (4.70)	44.83 (5.99)	87.50 (12.70)	87.36 (19.76)
Expressive Speech	33.00 (2.53)	35.20 (4.32)	48.75 (7.83)	78.58 (15.20)	84.13 (17.29)
Writing	48.18 (5.76)	49.10 (6.77)	55.42 (9.95)	83.83 (12.65)	79.75 (16.87)
Reading	39.73 (2.00)	41.80 (2.35)	49.00 (7.85)	64.75 (6.70)	68.75 (12.75)
Arithmetic	43.91 (2.88)	48.20 (10.05)	61.50 (12.62)	103.42 (2.59)	96.00 (19.13)

Table 5 (Continued)

Means and Standard Deviations for the Five Groups on the Luria-Nebraska Neuropsychological Battery

Tests	Groups				
Luria -Nebraska	NOR I	NOR II	DEP	AD	MID
Scales	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)
Memory	42.36 (6.09)	53.20 (10.44)	70.67 (8.36)	88.18 (2.59)	87.00 (5.66)
Intellectual Processes	37.82 (6.32)	42.60 (11.81)	58.05 (10.48)	83.00 (6.29)	84.13 (10.44)
Pathognomic	37.00 (7.91)	49.00 (9.26)	56.40 (5.68)	92.00 (14.80)	88.50 (12.44)
Left Hemis.	37.64 (1.86)	41.60 (6.54)	51.75 (4.71)	84.68 (10.41)	80.63 (11.67)
Right Hemis.	32.91 (3.83)	38.00 (6.53)	45.25 (14.15)	74.58 (8.32)	69.38 (10.14)

all the Luria-Nebraska scales have been presented in Table 6.

The analyses of the LNNB data reveal that the AD and MID subjects have considerable neuropsychological deficits compared to the normal healthy elderly (both age groups) and depressives. As can be seen in Table 5 and Figure 1, both AD and MID groups performed extremely poorly with mean T scores much above 60 on all the scales. One of the criteria of brain damage suggested by Golden and his associates (1980) is T scores above 60 on at least three scales. It is clear, therefore, that both Alzheimer's and Multi-infarct patients have pronounced neuropsychological defits in respect of their LNNB performance.

In the case of the depressed elderly, their LNNB scores were generally higher than the normal controls (both age groups). However, except for the memory score of T 70, most scaled scores were under T 60, indicating generally intact neuropsychological functioning in the depressed elderly.

When the two degenerative brain disease categories were compared with the depressive disorder, there were highly significant differences ($p < .05$) on all the LNNB scales (Table 6). The patterns of the scaled scores were also completely different (see the section on Pattern Analysis of LNNB scores). However, there were no significant differences between the AD and MID groups in any of the fourteen Luria-Nebraska scales. Also, their patterns of performance (indicating high and low points) were similar to each other (Figure 1).

The LNNB performance of the old and young normal elderly did not fall in the impaired range, since all their scaled scores were

Table 6

Analysis of Variance F Values and Newman-Keuls

Results of the LNNB Scales for the Five Groups

LNNB SCALES	F* VALUES	NEWMAN-KEULS RESULTS**
MOTOR	51.00	AD, MID, DEP > NOR I AD, MID > NOR II AD, MID > DEP
RHYTHM	53.08	AD, MID, DEP, NOR II > NOR I AD, MID, DEP > NOR II AD, MID > DEP
TACTILE	51.76	AD, MID, DEP > NOR I AD, MID > NOR II AD, MID > DEP
VISUAL	34.19	AD, MID, DEP > NOR I AD, MID, DEP > NOR II AD, MID > DEP
RECEPTIVE SPEECH	69.69	AD, MID, DEP > NOR I AD, MID > NOR II AD, MID > DEP
EXPRESSIVE SPEECH	50.18	AD, MID, DEP > NOR I AD, MID, DEP > NOR II AD, MID > DEP
WRITING	26.84	AD, MID > NOR I AD, MID > NOR II AD, MID > DEP
READING	34.99	AD, MID, DEP > NOR I AD, MID, DEP > NOR II AD, MID > DEP
ARITHMETIC	51.42	AD, MID, DEP > NOR I AD, MID, DEP > NOR II AD, MID > DEP
MEMORY	85.71	AD, MID, DEP, NOR II > NOR I AD, MID, DEP > NOR II AD, MID > DEP

Table 6 (Continued)

Analysis of Variance F Values and Newman-Keuls

Results of the LNNB Scales for the Five Groups

LNNB SCALES	F* VALUES	NEWMAN-KEULS RESULTS"
INTELLECTUAL PROCESSES	56.22	AD, MID, DEP > NOR I AD, MID, DEP > NOR II AD, MID > DEP
PATHOGNOMONIC	57.54	AD, MID, DEP, NOR II > NOR I AD, MID > NOR II AD, MID > DEP
RIGHT HEMISPHERE	86.85	AD, MID, DEP > NOR I AD, MID, DEP > NOR II AD, MID > DEP
LEFT HEMISPHERE	42.84	AD, MID, DEP > NOR I AD, MID, DEP > NOR II AD, MID > DEP

*ALL F VALUES ARE SIGNIFICANT AT .00005 LEVEL

"ALL COMPARISON GROUPS LISTED ARE SIGNIFICANT AT .05 LEVEL

NOTE - 1. GREATER THAN (>) MEANS POORER PERFORMANCE ON ALL THE SCALES
2. AD = ALZHEIMER'S DISEASE; MID = MULTI-INFARCT DEMENTIA;
DEP = DEPRESSION; NOR I = NORMAL (AGE 55-74);
NOR II = NORMAL (AGE 75 AND OVER)

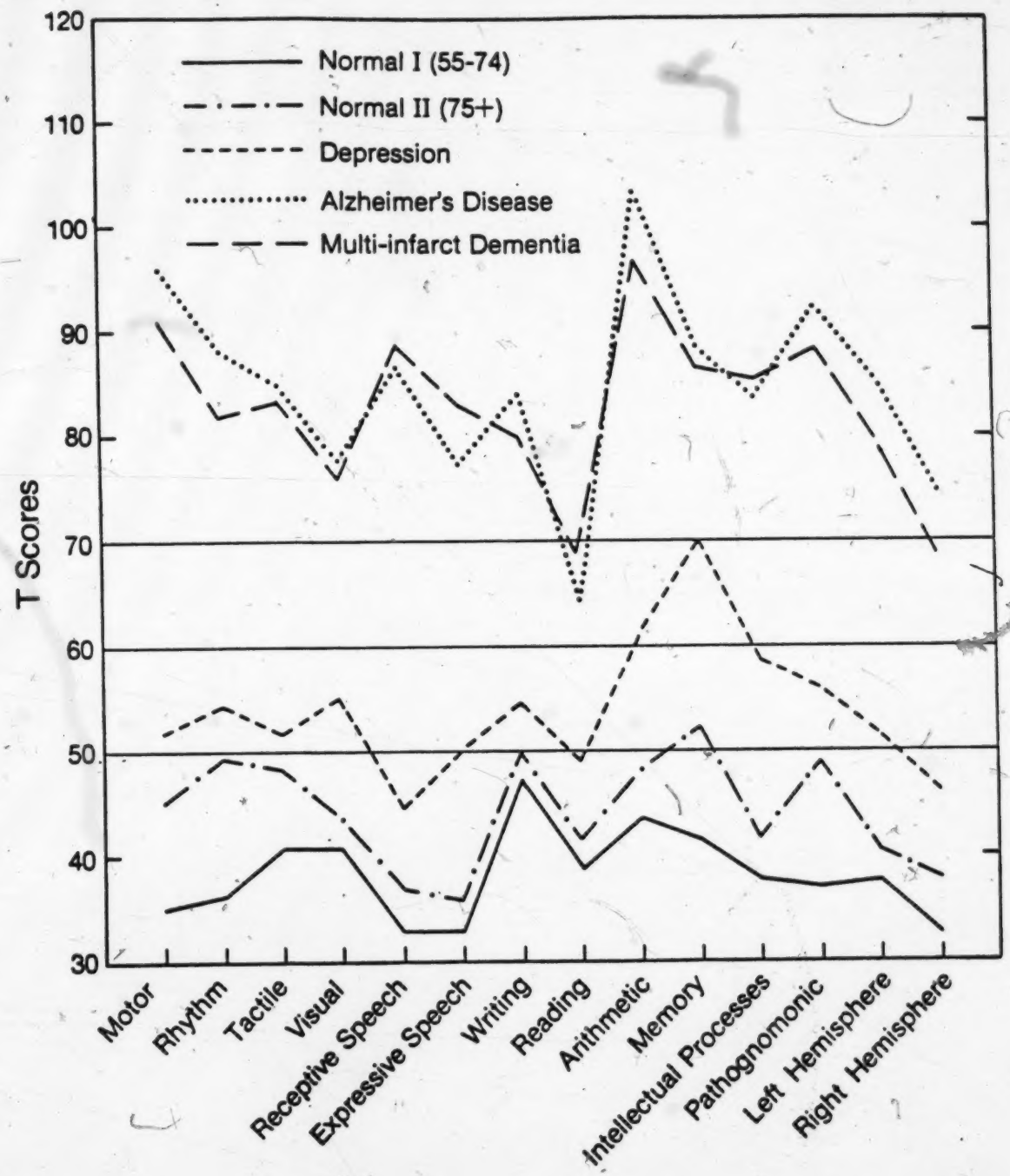

Figure 1. Luria-Nebraska profile summaries of the five groups. (Higher scores indicate poorer performance).

under T 55 (Figure 1). However, the old elderly scored significantly higher ($p < .05$) than the young elderly on the Motor, Rhythm, Memory, and Pathognomonic scales (Table 6).

The presence of brain damage on the LNNB is also evaluated on the basis of the number of elevated scales over the critical level determined by the formula suggested in the test Manual (Golden et al., 1980). The critical level is the highest score obtained by a person that can be considered normal with adjustment for age and educational level. According to Golden and his associates (1981), three or more elevated scales in a profile represent brain damage. As shown in Table 7, the average number of elevated scales was 10.4 for both the AD and MID groups suggesting considerable neuropsychological deficits among the dementia patients. (The number of elevated scales above the critical level for all subjects in each group was used to calculate the average, see Table 7.) In contrast, the depressive patients had only 1.3 elevated scales on the average indicating virtually no neuropsychological impairment. As expected, none of the LNNB clinical scales was elevated in the profile of the two normal control groups.

Pattern Analysis of LNNB Scores

The interpretive manual of the LNNB suggests the use of pattern analysis to obtain a global picture of the nature of brain damage, since simple elevations cannot be interpreted in isolation. As explained in the manual:

> The major and most useful approach of the battery is through an overall pattern analysis of the scales and items combined with additional information gained from the qualitative analysis of the test performance (Moses et al., 1983, p.54).

Thus, the LNNB scores of the five groups have been examined in terms

Table 7 .

Presence, Lateralization, and Localization of Brain Dysfunction on the Luria-Nebraska Scales for the Five Groups

Groups	Presence	Lateralization		Localization			
	No. of Elevated Scores on Clinical Profile	Localization Scales		No. of Elevated Scores on the Localization Profile	1-Pt. Code	2-Pt. Code	3-Pt. Code
		Highest	2nd Highest				
AD							
1	11	L	R	8			LPO/RPO/RF
2	11	L	R	8			LF/LPO/RPO
3	9	L	R	7			LF/LPO/RT
4	11	R	L	8			LPO/RPO/LT
5	12	L	R	7		LPO/RPO	
6	12	L	R	8			LF/LPO/RPO
7	11	L	L	8			LF/LPO/RPO
8	11	L	L	8			LF/LPO/RPO
9	11	L	R	8			LF/LPO/RPO
10	4	L	L			LPO/LT LF/RPO	
11	11	L	R	5			
12	11	R	L	8			RPO/LT/LF
	Mean = 10.4	No. of clear Lateralization = 3		Mean = 7.25	0	3	9

Table 7 (Continued)

Presence, Lateralization, and Localization of Brain Dysfunction

on the Luria-Nebraska Scales for the Five Groups

Groups	Presence	Lateralization			Localization		
	No. of Elevated Scores on Clinical Profile	Localization Scales		No. of Elevated Scores on the Localization Profile	1-Pt.Code	2-Pt. Code	3-Pt. Code
		Highest	2nd Highest				
MID							
1	12	R	L	8			RPO/LPO/LF
2	12	L	R	7		LPO/RPO	
3	11	L	R	8			RPO/LF/LPO
4	8	R		4	RPO		
5	12	L	R	8			LF/RPO/LPO
6	11	L	R	8			LF/RPO/LPO
7	11	L	-	8	LF		
8	6	R	-	1	RPO		
	Mean = 10.4	No. of clear Lateralization = 3		Mean = 6.5	3	1	4

Table 7 (Continued)

Presence, Lateralization, and Localization of Brain Dysfunction

on the Luria-Nebraska Scales for the Five Groups

Groups	Presence	Lateralization			Localization		
	No. of Elevated Scores on Clinical Profile	Localization Scales		No. of Elevated Scores on the Localization Profile	1-Pt. Code	2-Pt. Code	3-Pt. Code
		Highest	2nd Highest				
DEP							
1	2	L	-	1	LF		
2	2	-	-	-			
3	0	-	-	-			
4	0	-	-	-			
5	2	L		1	LPO		
6	1	-	-	-			
7	2	-	-	-			
8	1	-	-	-			
9	1	-	-	-			
10	1	R	-	1	RF		
11	2	-	-	-			
12	1	-	-	-			
	Mean = 1.3	No. of clear Lateralization = 3		Mean = 0.25	3	0	0

NOR I † NO SCORE IN ANY CATEGORY
NOR II †

of the (a) clinical, (b) lateralization, (c) localization, and (d) factor scale.

Profile of Clinical Scales. The results showed that the profile of scales for the two degenerative groups were essentially similar (Figure 1). The pattern of the means for the depressive group was similar to the pattern of the means for two normal groups except on the Arithmetic and Memory Scales.

Lateralization. The purpose of lateralization analysis is to identify the hemispheric involvement in neuropsychological impairments. Following the procedure recommended in the Interpretation Manual Vol. 1 (Moses et al., 1983), lateralization is determined by the method of localization scales. This method identifies the two highest T scores in order of their magnitude on the Localization Scale Profile. When the two highest scales are the same with respect to the hemisphere, lateralization is relatively unambiguous. However, in case of different hemispheres identified by the two scales, impairment is considered diffuse or bilateral.

As can be seen in Table 7, 25% of the AD patients present evidence of clear lateralization in the Left Hemisphere. However, the data suggest bilateral or diffuse damage in the majority of the AD patients. In the case of the MID patients, the data also suggest bilateral and diffuse hemispheric impairment (Table 7). Table 7 shows that the depressive group has very few scores above the critical level on the Localization Scales Profile suggesting virtually no hemispheric lesion. The normal controls also do not show any lateralization.

Localization. Localization analysis helps identify the locus of brain lesion. As suggested in the interpretive manual of LNNB

(Moses et al., 1983), eight localization scales were scored separately for each subject in order to identify the specific lesion areas in the brain. Most of the AD patients received a three-point code on the localization scale (see Table 7). The three-point code is assigned when three or more scales are above the critical level and within five points of one another. The mean scores of the AD group indicate high points on the Left Parietal-Occipital and the Right Parietal-Occipital Scales with secondary elevation on the Left Frontal Scale (Figure 2). This suggests bilateral and diffuse disorder. The MID group also had a similar localization profile with relative elevations on the same scales (Figure 2).

The depressives and normal controls had no elevation on any localization scale (Figure 2). This confirms the fact that these groups did not have impairment in any area of the brain.

Factor Scales. The factor scales are empirically derived from factor analysis of the items within each clinical scale of the Luria-Nebraska. The factor scales differ from clinical scales in that the former are comprised of heterogeneous sets of items derived from empirical data as well as clinical judgment. Thus, the factor scales were formed by the test authors (Golden et al., 1982) for the purpose of subdividing the clinical scales into subscales purely on an empirical basis. The factor scales allow useful information about the individual's performance and more clearly indicates the person's specific neuropsychological deficits (Golden et al., 1981).

As suggested by Golden et al. (1981), for a more objective and precise interpretation of the LNNB data of this study, factor scale profiles were prepared. Figure 3 presents the factor score profile of

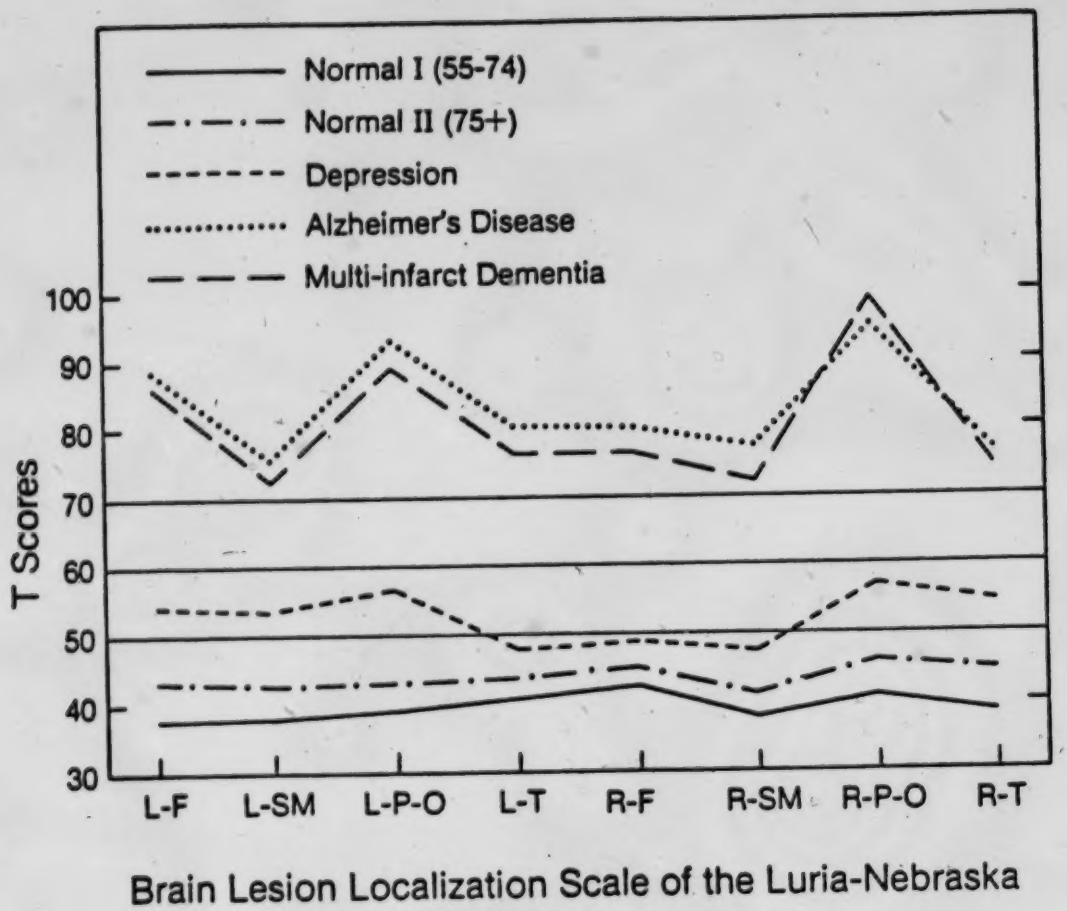

Figure 2. Mean localization score profile for the five groups. (Higher scores indicate poorer performance).

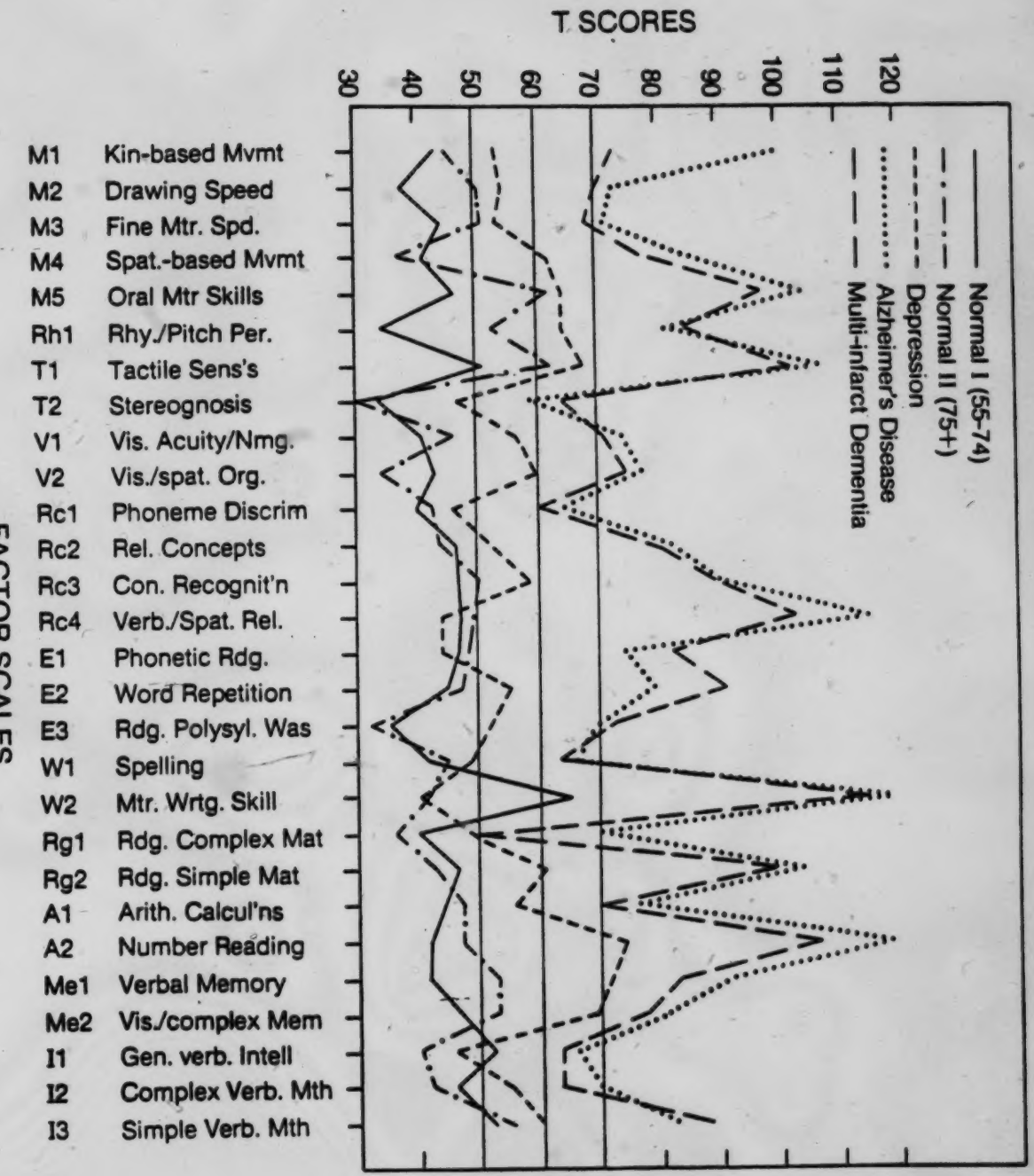

Figure 3. Mean factor scales profiles for the five groups. (Higher scores indicate poorer performance).

the five groups. The analysis of the factor scores indicates widespread impairment in the AD and MID patients. However, no specific impairment is noted in the control (NOR I & NOR II) and Depression (DEP) groups.

Other Neuropsychological Tests

Aphasia Test

The results in Table 8 and Figure 4 indicate that the normal group were vastly superior to the AD and MID groups on the Halstead Aphasia Screening Test. Whereas the average number of errors in the two normal groups was around three, the AD and MID groups made on the average 28.7 and 28.3 errors respectively (Table 8). The mean number of errors was also very low (5.75) in the depressive group. However, neither Normal I nor Normal II differed from the DEP group. Similarly, there was no significant difference between MID and AD patients.

Trail Making Test (Part A and Part B)

The results of the Trail Making Test--Part A (Table 9 and Figure 5) indicate that the AD, MID and DEP patients generally took a longer time to complete the task than the Normal I and Normal II groups (except that NOR II and DEP did not differ significantly). The depressive patients were better than both the AD and MID groups. However, no significant difference existed between AD and MID patients. Also, the two normal controls (NOR I and NOR II) did not differ significantly on this task.

With respect to the Trail Making--Part B (Table 9 and Figure 5), most group differences were significant ($p < .05$), similar to the

Table 8

Means, Standard Deviations, ANOVA F Values, and Newman-Keuls Results of the Aphasia Screening Test for the Five Groups

GROUPS	MEAN	SD	F	NEWMAN-KEULS RESULTS"
NORMAL I	1.91	1.45		
NORMAL II	3.00	2.79		AD, MID > NOR I
DEP	5.75	3.72	33.92*	AD, MID > NOR II
AD	28.67	9.15		AD, MID > DEP
MID	28.25	15.07		

NOTE - THE APHASIA SCORES ARE IN TERMS OF NUMBER OF ERRORS

*SIGNIFICANT AT .00005 LEVEL

"ALL COMPARISON GROUPS LISTED ARE SIGNIFICANT AT .05 LEVEL ($P < .05$)

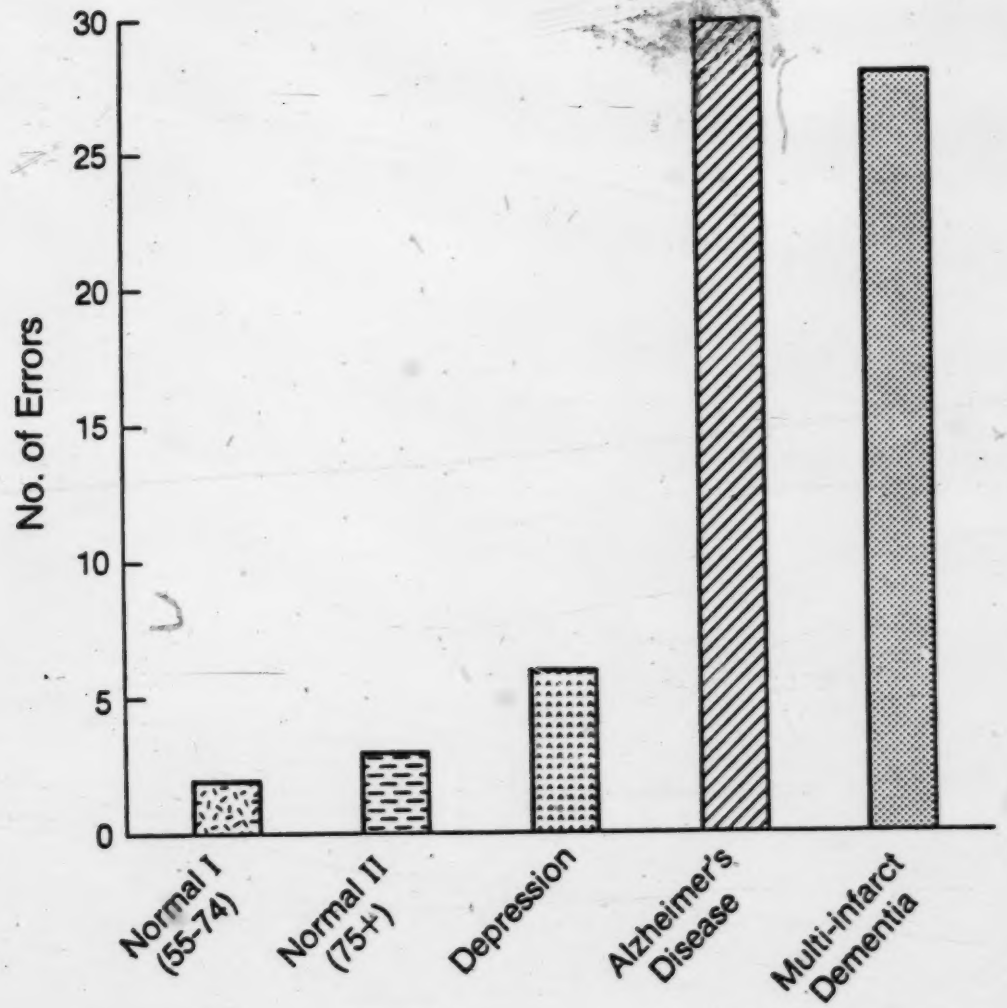

Figure 4. Aphasia Screening Test Scores of the five groups.

Table 9

Means, Standard Deviations, ANOVA F Values, and Newman-Keuls Results of the Trail Making Test for the Five Groups

GROUPS	MEAN	SD	F	NEWMAN-KEULS RESULTS"
TRAIL A				
NOR I	50.18	13.60		
NOR II	85.80	33.85	29.08*	AD, MID, DEP > NOR I AD, MID > NOR II AD, MID > DEP
DEP	103.75	39.40		
AD	195.00	65.44		
MID	217.50	38.08		
TRAIL B				
NOR I	76.73	23.95		
NOR II	128.50	39.86	47.97*	AD, MID, DEP, NOR II > NOR I AD, MID > NOR II AD, MID > DEP
DEP	166.40	66.42		
AD	312.33	48.17		
MID	295.00	52.37		

NOTE - THE TRAIL MAKING SCORES FOR BOTH A & B ARE IN TERMS OF TIME TAKEN (IN SECONDS)

*SIGNIFICANT AT .00005 LEVEL

"ALL COMPARISON GROUPS LISTED ARE SIGNIFICANT AT .05 LEVEL

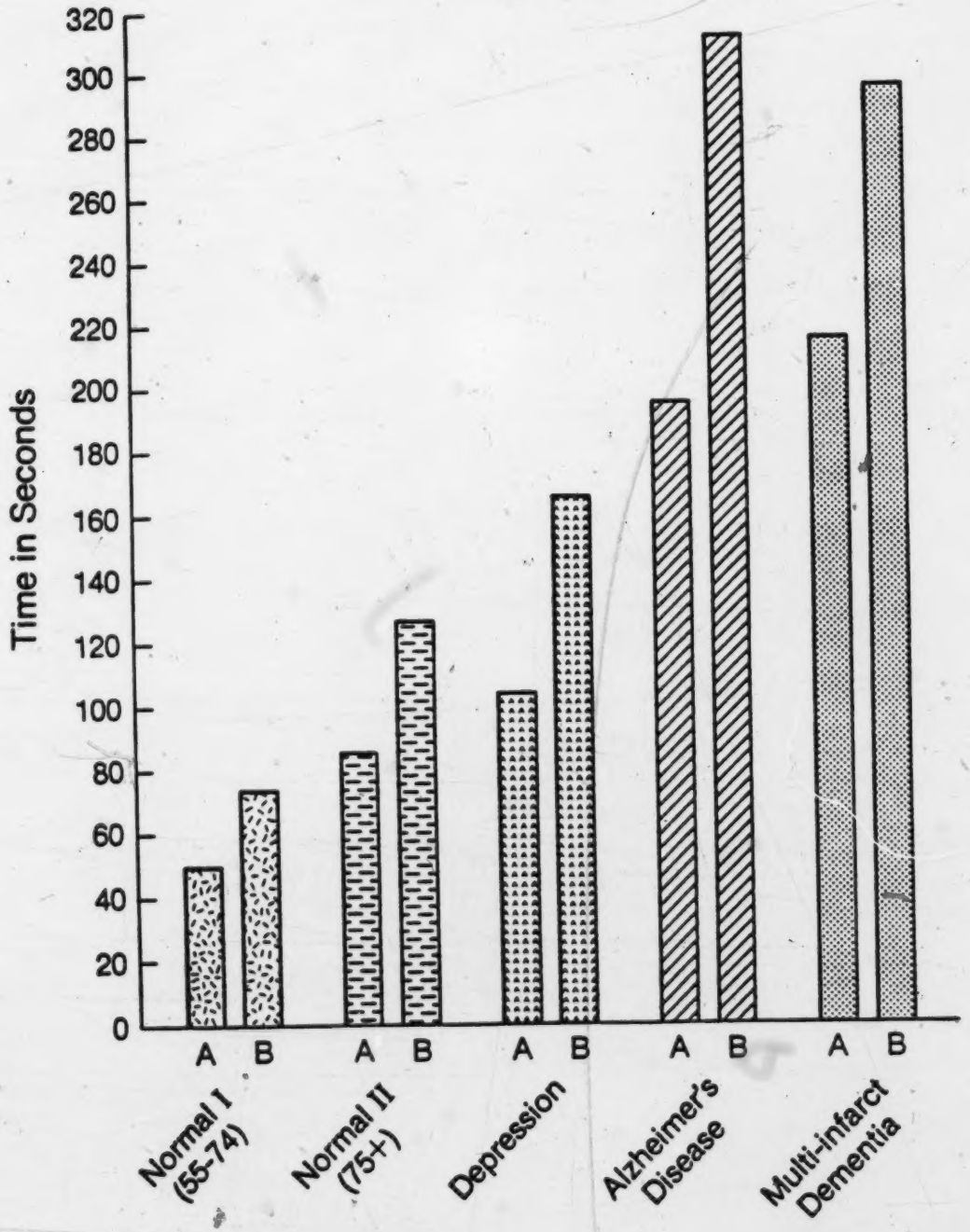

Figure 5. Trail Making Test (A & B) Scores of the five groups. (Shorter time indicates better performance).

results obtained in Part A of the TMT. The young elderly (NOR I) performed much better than the other patient groups. Unlike Part A, the young elderly (NOR I) were significantly ($p < .05$) better than the old elderly (NOR II) on this task. Again, just like Part A, the old elderly (NOR II) demonstrated superior ability to the AD and MID patients. Depressives were better than both AD and MID, but no significant difference existed between AD and MID.

Wechsler Memory Scale

Means, standard deviations, F ratios and associated probabilities of the Wechsler Memory Scores for the five groups are presented in Table 10 and Figure 6. The group differences were highly significant ($p < .05$) in respect of all the eight Wechsler Memory scores. Generally speaking, the AD and MID patients performed worse on all the subtests than the other three groups. Newman-Keuls multiple comparisons of group means (see Table 11) reveals that although the two normal controls (NOR I and NOR II) were better than the AD and MID groups in respect of the Information, Orientation and Mental Control tests, they did not differ significantly from the depressive group on these tests. However, the depressive group performed poorly in comparison with the Normal I and Normal II groups on the Logical Memory, Digit Total and Total score responses. The depressives were better than both AD and MID groups on all Wechsler Memory scores.

It is interesting to note that there were no significant differences between the young and old elderly on Information, Orientation, Mental Control, Logical Memory and Digit Total tests. But the young elderly (NOR I) were significantly ($p < .05$) better than

Table 10

Means, Standard Deviation, F ratio, and Significance Levels for the Five Groups on the Wechsler Memory Scale

TESTS	NOR I	NOR II	DEP	AD	MID	F*
Wechsler Memory	M (SD)	M (SD)	M (SD)	M (SD)	M (SD)	
Info.	6.00 (0.00)	6.00 (0.00)	5.08 (1.51)	2.08 (1.88)	2.63 (1.92)	19.93
Orient.	5.00 (0.00)	5.00 (0.00)	4.67 (0.89)	2.58 (1.08)	3.00 (1.96)	22.08
Mental Control	8.00 (1.19)	7.90 (2.28)	6.58 (2.07)	2.58 (2.47)	1.88 (2.23)	19.52
Logical Memory	13.36 (1.16)	11.55 (2.61)	6.88 (1.25)	1.25 (1.90)	3.19 (3.15)	69.34
Digit Total	11.63 (1.21)	11.10 (1.10)	9.42 (1.66)	5.67 (2.54)	5.25 (1.49)	34.54
Visual Repro	8.18 (2.89)	4.00 (2.45)	3.42 (2.54)	0.50 (0.79)	0.88 (1.36)	21.37
Assoc. Learning	15.96 (2.71)	12.00 (2.56)	12.45 (4.61)	4.29 (1.71)	4.50 (2.30)	35.16
Total Score	68.23 (6.58)	57.55 (6.61)	47.33 (11.36)	18.96 (7.82)	21.36 (10.56)	

NOTE - THE HIGHER THE SCORE, THE BETTER THE PERFORMANCE

*SIGNIFICANT AT .00005 LEVEL

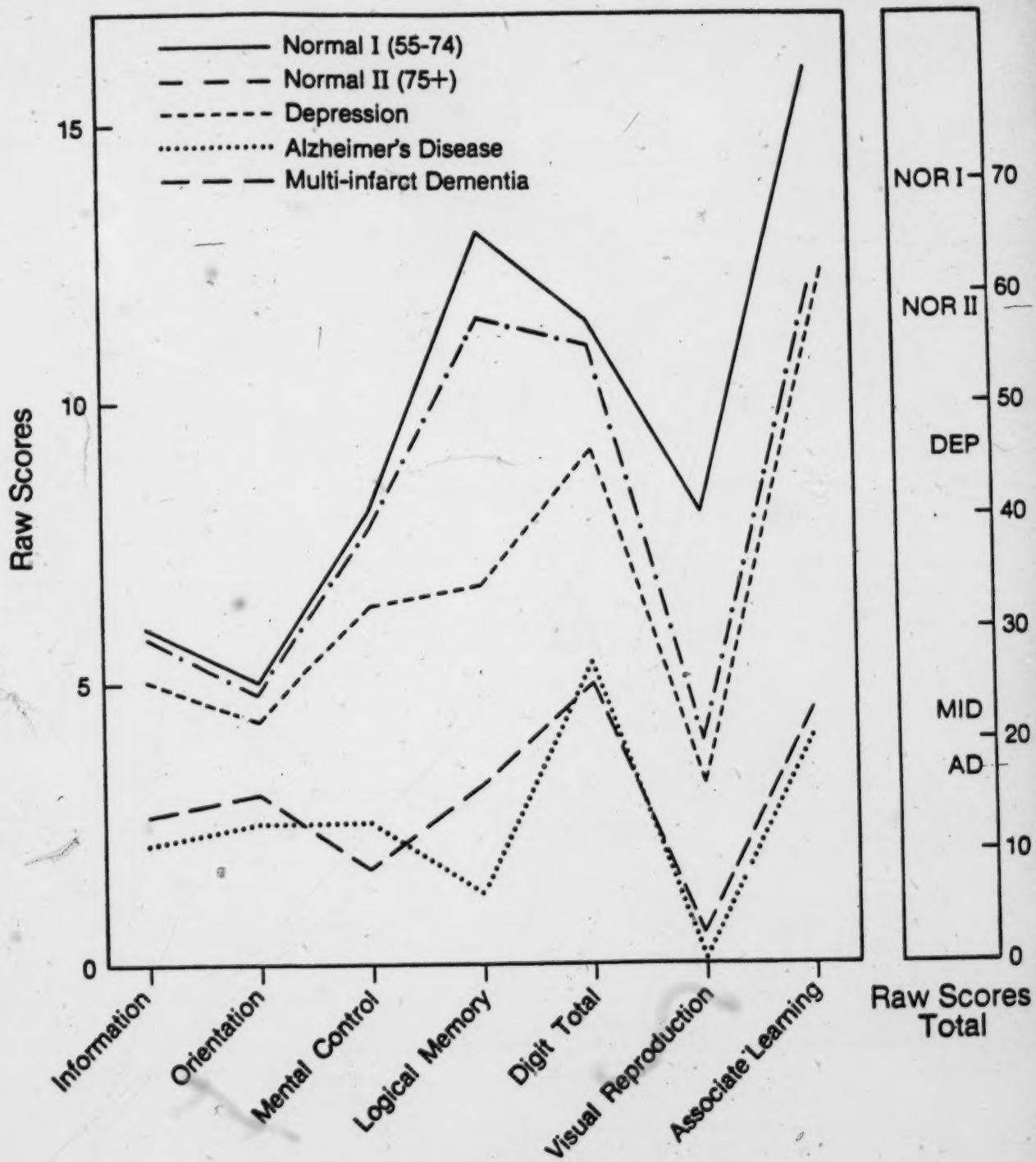

Figure 6. Wechsler Memory Scale Scores of the five groups. (Higher scores indicate better performance).

Table 11

Analysis of Variance F Values and Newman-Keuls Results

of the Wechsler Memory Scale for the Five Groups

WMS TESTS	F VALUE	"NEWMAN-KEULS RESULTS"
INFORMATION	19.93*	AD, MID < NOR I AD, MID < NOR II AD, MID < DEP
ORIENTATION	22.08*	AD, MID < NOR I AD, MID < NOR II AD, MID < DEP
MENTAL CONTROL	19.52*	AD, MID < NOR I AD, MID < NOR II AD, MID < DEP
LOGICAL MEMORY	69.34*	AD, MID, DEP < NOR I AD, MID, DEP < NOR II AD, MID < DEP
DIGIT CONTROL	34.54*	AD, MID, DEP < NOR I AD, MID, DEP < NOR II AD, MID < DEP
VISUAL REPRODUCTION	21.37*	AD, MID, DEP, NOR II < NOR I AD, MID < NOR II AD, MID < DEP
ASSOCIATE LEARNING	35.16*	AD, MID, DEP, NOR II < NOR I AD, MID < NOR II AD, MID < DEP
TOTAL SCORE	64.73*	AD, MID, DEP, NOR II < NOR I AD, MID, DEP < NOR II AD, MID < DEP

NOTE - *SIGNIFICANT AT .00005 LEVEL

"ALL COMPARISON GROUPS LISTED ARE SIGNIFICANT AT .05 LEVEL

the old elderly (NOR II) on three tests, namely, Visual Reproduction, Associate Learning and Total Score.

Wisconsin Card Sorting Test

Table 12 and Figure 7 summarize the performance of the five groups on the Wisconsin Card Sorting Test. The analyses of variance showed that the five groups were significantly ($p < .05$) different on all the nine Wisconsin scores except on the "Failure to maintain set" score. The performances of the AD, MID and depressive groups were significantly impaired on the eight Wisconsin measures when compared to the normal controls (Table 13). One of the important measures of brain impairment on the Wisconsin Card Sorting Test is the number of Perseverative Responses made by the subjects. The greater the number of errors, the more severe is the brain impairment. According to Robinson et al. (1980), a Perseverative Response score of 19 or more should be regarded as indicative of brain damage. Both the AD and the MID groups obtained extremely high (62.33 and 61.75) scores on the "Perseverative Responses" measure suggesting the presence of considerable brain impairment. The depressed elderly group had a mean score of 28.50 indicating some organic involvement.

Another index of brain damage on the Wisconsin Card Sorting is the score on "Categories Achieved" (ten consecutive correct responses). The present data indicate that AD and MID groups are the worst performers on this measure. On the average the AD patients achieved 1.08 categories and the MID patients 1. The number of categories achieved by the normal controls was between 5 and 6, while the mean score for the depressive is 4.3. When the "Total Errors" score is examined, a similar discrepancy was present in the

Table 12

Means and Standard Deviations for the Five Groups on the Wisconsin Card Sorting Test

	NORMAL I (N = 11) Mean (SD)	NORMAL II (N = 10) Mean (SD)	DEP (N = 12) Mean (SD)	AD (N = 12) Mean (SD)	MID (N = 8) Mean (SD)
Category Achieved	5.91 (0.30)	5.20 (0.92)	4.33 (1.07)	1.08 (0.79)	1.00 (1.07)
Total Errors	27.23 (6.33)	35.70 (9.03)	43.42 (10.49)	75.50 (4.66)	77.63 (10.54)
Perseverative Errors	14.91 (5.39)	21.10 (5.90)	22.08 (7.13)	48.42 (15.50)	51.86 (17.52)
% Perseverative Errors	26.29 (26.94)	17.57 (4.00)	17.23 (5.56)	38.01 (12.20)	40.51 (13.66)
NonPerseverative Errors	13.55 (6.24)	14.60 (5.17)	22.83 (6.90)	27.08 (14.15)	25.75 (9.75)
Perseverative Responses	16.18 (5.93)	26.00 (8.64)	28.50 (6.40)	62.33 (21.35)	61.75 (20.39)
Trials to 1st Category	11.18 (11.66)	10.30 (17.20)	17.08 (17.13)	56.50 (43.45)	69.63 (49.36)
% Conceptual Level Responses	69.66 (4.80)	65.17 (6.26)	53.03 (9.72)	29.93 (7.37)	25.19 (11.87)
Failure to Maintain Set	1.55 (1.23)	2.30 (1.57)	2.25 (1.45)	1.58 (0.79)	2.25 (1.28)

NOTE - EXCEPT FOR "CATEGORY ACHIEVED" AND "% CONCEPTUAL LEVEL RESPONSES" WHERE LOWER SCORES INDICATE IMPAIRMENT, IN ALL OTHER TESTS HIGHER SCORES INDICATE IMPAIRED FUNCTIONING.

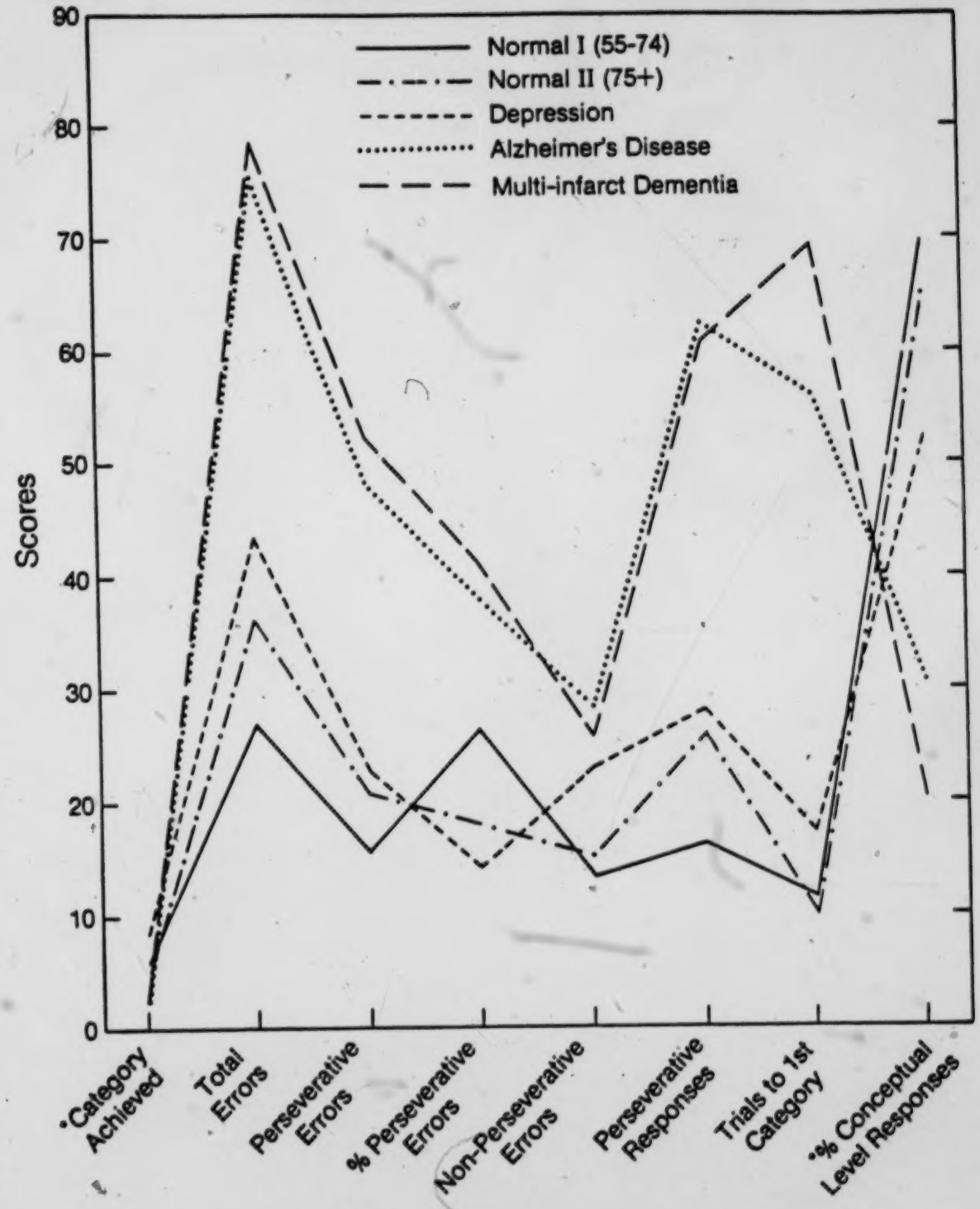

Figure 7. Wisconsin Card Sorting Test Scores of the five groups. (Higher scores indicate poorer performance on all categories except 'Category Achieved' and '% Conceptual Level Responses' where higher scores indicate better performance).

Table 13

Analysis of Variance F Values and Newman-Keuls Results of the Wisconsin Card Sorting Test for the Five Groups

WCST	F VALUE	NEWMAN-KEULS RESULTS"
CATEGORY ACHIEVED	73.40*	AD, MID, DEP < NOR I AD, MID, DEP < NOR II AD, MID < DEP
TOTAL ERRORS	78.03*	AD, MID, DEP, NOR II > NOR I AD, MID, DEP > NOR II AD, MID > DEP
PERSEVERATIVE ERRORS	24.00*	AD, MID, DEP > NOR I AD, MID > NOR II AD, MID > DEP
% PERSEVERATIVE ERRORS	5.56*	AD, MID, DEP > NOR I AD, MID > NOR II AD, MID > DEP
NON PERSEVERATIVE ERRORS	5.02*	AD, MID, DEP > NOR I AD, MID, DEP > NOR II
PERSEVERATIVE RESPONSES	24.64*	AD, MID > NOR I AD, MID > NOR II AD, MID > DEP
TRIALS TO 1ST CATEGORY	8.66*	AD, MID > NOR I AD, MID > NOR II AD, MID > DEP
% CONCEPTUAL LEVEL RESPONSES	61.35*	AD, MID, DEP < NOR I AD, MID, DEP < NOR II AD, MID < DEP
FAILURE TO MAINTAIN SET	2.18	NOT SIGNIFICANT

NOTE - *SIGNIFICANT AT .001 LEVEL

"ALL COMPARISON GROUPS LISTED ARE SIGNIFICANT AT .05 LEVEL

pathological groups. The AD and MID groups have more than twice the number of "Total Errors" obtained by the normal control groups. Even the depressed group had a substantially poor score on the Total Errors measure (Table 12).

In summary, the young elderly performed better than all other groups, and the depressives were superior to the Alzheimer's and Multi-infarct groups. Although the normal old elderly were better than the Alzheimer's and Multi-infarct subjects, this group was not always superior to the depressive group. Specifically, there were no significant differences between the older normals and depressive groups on Perseverative Errors, % Perseverative Errors, Perseverative Responses, and Trials to 1st Category. With the exception of the Total Errors score, there were no significant differences between the young and old elderly on the Wisconsin Card Sorting Test.

CHAPTER V

DISCUSSION AND CONCLUSIONS

This final chapter will highlight the main findings, and offer interpretations. It will focus first on the main hypothesis, and then on different aspects of the relationships at issue. The effectiveness of the battery of tests used in differentiating between AD and MID on the one hand and Depression on the other will be underlined. The special problem of distinguishing between AD and MID will be discussed in the light of theoretical issues as well as limitations of diagnostic instruments. Finally, directions for future research will be discussed.

The main hypothesis predicted that significant neuropsychological impairment would be found in the elderly with Alzheimer's Disease (AD), Multi-infarct Dementia (MID) and Depression (DEP) compared to the normal, healthy elderly. The results of the present study strongly confirmed this hypothesis. AD and MID patients performed consistently worse on all neuropsychological measures. On the LNNB scales, almost all scores of these two groups were between ten to thirty points above their critical level revealing comprehensive deficits in neuropsychological functioning. Interestingly enough, the worst performance of both AD and MID group was on the Arithmetic Scale followed by the Motor Scale. According to the manual (1980) elevated scores on the Arithmetic scale are indicative of lesions in all parts of the brain with specific involvement of the Left parietal-occipital area. Behaviorally, poor performance on the

Arithmetic Scale reflects deficiency in attention and concentration, verbal reasoning, memory, flexibility, and sequencing ability. The Motor Scale suggests deficits of simple to complex sensory-motor function which are likely to be affected adversely by impairment in several areas of the brain, such as optical-spatial, verbal, kinesthetic and kinetic areas. However, according to Luria (1973), multiple cortical systems must work together for normal motor movements.

The third highest score of both the AD and MID groups was on the Pathognomic Scale. Golden et al. (1982b) assert that marked elevations of the Pathognomic Scale reveal acute destructive lesions. This inference is consistent with the clinical observations of the Alzheimer's and Multi-infarct patients.

Other elevated scores of the AD and MID patients were on the Rhythm, Receptive Speech, Memory and Intellectual Process Scales. The Rhythm Scale measures the ability to recognize and recall auditory information in a sequential pattern. According to Golden and his associates (1982b), attention and concentration abilities play major roles in this scale. Brain impaired and emotionally disturbed persons generally perform very poorly. Receptive Speech is concerned with phonemic hearing, word comprehension, simple sentence comprehension and logical grammatical structures. Again, Golden and his associates (1982) suggest that high scores on this scale are most likely due to damage to the left hemisphere. Poor performance on the Memory Scale indicates difficulties with retention, retrieval and other aspects of memory including attention, concentration and mental flexibility. Extremely poor performance suggests frontal lobe damage, and either

hemisphere or bilateral dysfunction. Assessment of sequencing, problem solving and abstraction is made through the Intellectual Process Scale. As shown in Figure 1 and Table 5 both the AD and MID patients obtained relatively high scores indicating prefrontal lobe dysfunction in both hemispheres.

Analyses of the LNNB performance in terms of the localization and factor score profiles also support the conclusion that both AD and MID patients were greatly inferior to the depressives and normal controls (Figures 2, 3). No significant differences were found between depressives and normals on the localization and factor scales.

In respect of other neuropsychological measures, namely the Aphasia Screening Test, Trial Making Test (Part A and Part B), Wechsler Memory Scale and Wisconsin Card Sorting Test, the performance of the Alzheimer's and Multi-infarct elderly was extremely poor compared to the normal healthy elderly. These results are consistent with the performance on the Luria-Nebraska, and are supported by other research (Bigler, 1981; Brinkman et al., 1983; Schludermann et al., 1983). Particular deficiencies of the AD and MID patients have been revealed on the Visual Reproduction test of the Wechsler Memory Scale. Further deficiency in the AD patients is noted on the Logical Memory, whereas Mental Control is relatively weak in the MID group. Similar findings about the AD patients' performance on the Wechsler Memory Scale have been reported by Botwinick, Storandt, and Berg (1986) in their logitudinal study.

In case of the Depression group, its performance was also poor on all LNNB scales (except on the Tactile Scale where there was no significant difference) in comparison with the young elderly (NOR I,

55-74). Lack of significant difference between the depressives (DEP) and the young elderly (NOR I) on the Tactile Scale suggests that simultaneous processing of somatosensory impulses is not affected adversely in depressive disorder.

Compared to the normal young elderly, the depressives also performed poorly on the Trail Making Test (Part A and Part B), Wechsler Memory Scale, and Wisconsin Card Sorting Test. It may be recalled that the Trail Making Test measures visual conceptual, and visuo-motor tracking involving motor speed and attention functions which are highly sensitive to brain injury (Lezak, 1983; Goldstein, 1984). Performance on the Wechsler Memory Scale suggests that the depressives have impairment in a wide variety of memory functions. This is also consistent with the fact that the only elevated scale over their critical level on Luria-Nebraska is the Memory Scale. Impaired memory in depression has also been observed in many previous research studies (Arenberg, 1977, 1982; Gilleard, 1980; Kendrick, 1967; La Rue et al., 1986). On the Wisconsin Card Sorting Test, the depressives performed more poorly than the young elderly controls in respect of the "Category Achieved", "Total Errors", "Nonperseverative Errors" and "% of Conceptual Level Responses". Since none of the scores of the Depression (DEP) group was above the critical level no valid inference about brain injury can be made.

In this connection it may be noted that although the depressives performed significantly more poorly on most LNNB scales (except one) all scores are below their critical level indicating no neuropsychological deficit. The performance of the depressives on these neuropsychological tests supports clinical observations and

empirical findings that there is no organic involvement in depressive disorder (Blazer, 1982; Donnelly et al., 1982; Freidman, 1964; Wells, 1980).

The results of this study also revealed that there were some significant differences between the old elderly control and the Depression group on all the five neuropsychological tests used in this study. However, there did not appear to be any clear-cut pattern of differences in the neuropsychological functioning of these two groups. Examination of the results suggests that the depressives and old elderly normals generally show similar kinds of psychomotor retardation. But it must be acknowledged that in spite of their lower scores, the depressives and the old normal elderly score well within the normal range of neuropsychological functioning.

Contrary to the first expectation that the Alzheimer patients would show marked global neuropsychological impairment compared to the Multi-infarct patients who would show patchy or focal impairment, the present study did not find any significant difference between the two groups with regard to their neuropsychological performance. In fact both AD and MID groups obtained almost similar scores on all the measures used in this study.

The AD and MID patients produced similar profile patterns for the fourteen Luria-Nebraska clinical scales, suggesting no difference in their neuropsychological deficits. The localization score profiles also failed to show any difference between the AD and MID groups. The Localization Scale of the AD and MID groups present evidence of both diffuse and bilateral impairment. All localization scales were above their critical levels. The Localization Score profiles show that both

the AD and MID patients obtained the highest scores on the Left Parietal-Occipital, Right Parietal-Occipital and Left Frontal Scales. This suggests diffuse bilateral rather than focal impairment.

The Factor Score profiles of the Luria-Nebraska failed to show any difference between the AD and MID groups. It is interesting to note that although both groups have scored well above their critical levels on a majority of factor scales, the highest elevations are shown on the same scales in both groups.

Analysis of the Factor Scale profiles of the AD and MID groups showed that both groups scored above T 90 on items requiring motor movement, oral and kinesthetic, verbal and nonverbal skills, visuo-motor spatial ability and short-term memory. It should be further noted that these tasks require attention and concentration abilities which appear to be severely damaged.

According to the literature (Gustafson, 1985; Lauter, 1985) localization of neuropsychological impairment is an important dimension for making differential diagnosis of Multi-infarct Dementia. Some clinical reports claim that the deficits in MID patients are fluctuating, asymmetrical, stepwise and focal (Hachinski, 1974; Roth, 1978). In contrast, gradual and diffuse organicity is the distinguishing feature of Alzheimer's Disease, seen as indicating that the impairment is not confined to any specific areas of the brain. According to some research, due to the unpredictable and multifocal nature of the disease, there is no definite pattern of neuro-psychological deficits in Multi-infarct Dementia, and the extent of individual differences in the configuration of test scores is much larger in MID groups than in other clinical groups (Fuld, 1983;

Reisberg, 1983).

It is apparent that the present study was unable to indicate any distinctive pattern of performance in the AD and MID groups on the selected neuropsychological tests. This contradicts the findings of Perez and associates (1976) who were able to identify AD and MID patients on the basis of their WAIS performance 75% of the time and 100% of the time on the basis of their performance on the Wechsler Memory Scale. Similar findings with WAIS are reported in recent as well as earlier studies (Brinkman, 1984; Hopkins, 1953; Robin, 1945). However, some researchers suggest that behavioral manifestations in Multi-infarct Dementia can be indistinguishable from Alzheimer's Disease (Torack, 1978). Several other studies on various psychological and neuropsychological measures have also failed to demonstrate any significant difference between AD and MID patients (Grossi & Orsini, 1978; Schindler et al., 1984). A recent study on neuropsychological differentiation between Alzheimer's Disease and vascular dementia by Erkinjuntti, Laaksonen, Sulkava, Syrjalainen, and Palo (1986) also reports no specific pattern of impairment associated with the AD and MID. Cognitive decline was similar in both conditions. La Rue, Dessonville and Jarvik (1985), and Liston and La Rue (1986) have emphasized the fact that from the behavioral standpoint, it is difficult to operationalize such criteria as "patchy" deficits or "stepwise" deterioration. Thus it is not surprising to find lack of differentiation in the cognitive functioning of the patients suffering from degenerative brain diseases.

At this point it may be useful to look also at the distinctions made by some researchers between cortical and subcortical dementia.

During the past ten years some researchers have explained the differential nature of dementia in AD, MID and depression in terms of the concept of "subcortical" and "cortical" dementia (Albert, 1984; Benson, 1983; Cummings & Benson, 1984). Cortical dementia is characterized by difficulties in language, perception and praxia -- typically present in Alzheimer's Disease. On the other hand, subcortical dementia is commonly observed in Multi-infarct Dementia where slowness of intellectual effort, mood disturbance and apathy are major symptoms without aphasia, agnosia and apraxia. The findings of the present study do not support this conceptual scheme of classifying dementia into two major categories.

A number of recent studies have questioned the value of using vague anatomical terms like cortical and subcortical dementia to label clinical syndromes. As a matter of fact, pathological studies reveal that cortical and subcortical changes are present in most dementia patients (Herzog & Kemper, 1980; Perry, Tomlinsen, Blessed, et al., 1981; Tagliavini & Pilleri, 1983). In a recent review article on this topic, Whitehouse (1986) argues that the concept of cortical and subcortical dementia is not only ambiguous but has not been supported by well-designed experiments or clinical research. For example, Whitehouse (1986) explains:

> Cognitive dysfunction in different diseases may change at different rates. In early stages of Alzheimer's disease, memory alone may be impaired; later, language and perception may deteriorate. Does this suggest that patients with Alzheimer's disease evolve from a subcortical to a cortical dementia? (p.2)

He further explains:

> The use of the terms subcortical and cortical tends to emphasize the independence of these regions of brain, whereas in fact the importance of dense interconnections

between cortical and subcortical structures has suggested several models of dementing conditions.

Thus, on the basis of the psychological, pathological, radiological (e.g., PET) and neurochemical studies Whitehouse (1986) concluded that the validitiy of the distinction between cortical and subcortical dementia has not been established. This would be congruent with the results of the present study.

However, it may be premature to suggest that MID patients have diffuse bilateral impairment as suggested by the results of this research. A major issue is diagnosis of MID and AD that is recognized by all researchers. The available test and medical procedures are not completely reliable due to imprecise understanding of the nature of degenerative brain disease. Even autopsy does not provide definite identification because of the complex nature of brain-behavior relationships. Another problem is the lack of agreement among researchers regarding paradigms and models of brain functioning. The interconnectedness between different parts of the brain makes inference about localization of damage extremely difficult. The available neuropsychological tests may not be sensitive enough to detect the precise function of different brain regions (Liston & La Rue, 1986; Whitehouse, 1986).

Although no difference was found between the AD and MID patients on any neuropsychological test used in this research, the clinical observations made during the testing sessions do indicate behavioral differences between the two groups. In general, MID patients appeared to be more aware of their deficits compared to the AD patients. They showed considerable anger and frustration when they could not perform a task successfully. The MID patients also had much

greater mood fluctuations (ranging from cooperative to resentment) between different testing sessions. On the other hand, the AD patients were generally unaware of their lack of success on the test items. Even when apparently frustrated, they did not show any anger or resentment. There was a clear tendency on the part of the AD patients to make regressive statements, such as talking about childhood events most of the time. In some cases, the AD patients even manifested child-like behavior, such as giggling and dependence. Also, the feelings of insecurity and uncertainty were pronounced in the AD patients. In general, the AD patients were weepy, nervous, but compliant. However, it should be noted that both the AD and MID patients responded positively to gentle persuasion and the affectionate attitude of this examiner.

The second expectation, namely that the depressed elderly would perform significantly better on all neuropsychological tests compared to the Alzheimer and Multi-infarct patients was strongly supported by the results of this study (Tables 5-13, Figures 1-7). In comparison with the Depression group, both the AD and MID groups performed significantly worse on all neuropsychological tests used in this study. The profiles of the AD and MID groups clearly fell in the brain-impaired range, while the profile of the depressives was well within the normal range. As reported earlier, none of the obtained scores of the depressed patients on any neuropsychological tests was in the brain-impaired range.

Other studies have also shown that dementia patients obtain lower scores than do age-matched depressed and normal persons on most tests of cognitive functions (Donnelly & Murphy, 1982; La Rue et al,

1985; Wells, 1983). In their recent study of demented, depressive and normal healthy aged persons, La Rue et al. (1986) found that depressed and normal healthy subjects made only one error in associate learning tasks, whereas multiple errors were made by the dementia group.

However, the findings of the present study contradict Folstein and McHug (1978) who have demonstrated considerable cognitive losses in depressive patients. They believe that cognitive losses are significant enough to be called "the dementia syndrome of depression". Weingartner and associates (1981) found both qualitative and quantitative changes in information processing episodes among the depressive middle aged subjects. Others have also reported depressed older adults to score in the organic range on various memory tests (Gilleard & Pattie, 1980; Kendrick, 1967).

In view of the contradictory findings further research with increased sample size and varying chronicity of dementia and depression may provide more consistent information about the nature of neuropsychological impairment in these disorders.

Another expectation of this study was that there would be no significant difference in neuropsychological functioning between normal, healthy, young elderly (55 to 74 years) and normal, healthy, old elderly (75 years and older). The results of the present research indicate that by and large the groups were similar on the tests used. Both groups performed well within their critical levels on the Luria-Nebraska suggesting completely intact neuropsychological functioning even in old age. Performance on the other tests also supports the expectation of unimpaired cognitive functioning in normal young and old elderly.

However, the young healthy elderly have performed better on all those neuropsychological tests which measure visual-motor sequencing, psychomotor activites, particularly with speed-related tasks, auditory comprehension and short-term memory, including visual and auditory memory (Tables 5-13). The selective age-related cognitive changes manifested on the LNNB scales and other neuropsychological measures used in this study are in accordance with the findings of MacInness et al. (1980) and Erkinjuntti et al. (1986). They also reported gradual decline in memory and psychomotor functions with increasing age. However, these age-related cognitive changes in normal aging did not have any serious effect on their social functions and orientation to time, place and personal needs. The familial and automatic tasks such as reading, writing and expressive speech remained intact. It should also be emphasized that the above-mentioned differences between the young and old elderly are all considerably below the brain-impaired range. These results are quite consistent with other research reports indicating that healthy older people do not show any significant decline even in their ninth decade (Arenberg, 1982a; Park et al., 1984; Park & Puglisi, 1986).

It is evident that even though the present study used major neuropsychological tests, it failed to provide any specific neuropsychological pattern for dementia of different etiology (i.e., AD and MID). However, it was able to clearly differentiate between dementia and depression groups. The findings of this research have considerable clinical implications for making differential diagnosis which is essential for adequate treatment and rehabilitation programs. The test batteries were also successful in assessing cognitive decline

associated with advancing age. These age-related cognitive changes are important, in the assessment of degree of dementia and suspected early dementia. However, in view of the limited number of subjects in each group, the interpretive conclusion regarding cognitive decline with advancing age should be made with caution. Also more subjects are needed in both AD and MID groups before making any clinical assertions about the LNNB's usefulness in distinguishing between AD and MID.

In summary, the present research was concerned with the problem of brain-behavior relationship. The goal of a neuropsychological test battery is to establish the relationship between behavioral dysfunctions and corresponding brain lesions. For this purpose, it is necessary to cross-validate a neuropsychological test battery on clinical populations with relatively established brain lesions. In the present research two degenerative brain diseases and one psychiatric disorder were selected for this purpose. The results generally support Luria's notion that the test behavior is dependent upon the coordinated activity of various cortical and subcortical structures (Luria, 1980). The lack of any difference between AD and MID groups may be explained as due to overlapping cortical and subcortical functions producing diffuse impairment in both rather than due to any psychometric weakness of the assessment device.

Regarding brain-behavior relationship, Lezak (1983) explains that highly interconnected parts of the brain make it difficult to clearly specify a lesion to a particular area of the brain. All brain structures are not affected equally even in diffuse brain lesions. Similarly, all focal damages have some diffuse repercussions

(Goodglass, 1973; Smith, 1975; Teuber, 1969). Smith (1975, 1979) drew attention to the fact that precise localization of a brain lesion is virtually impossible from test behavior. Primary focal lesions in specified brain areas are found in very few medical conditions. Identification of specific lesions also depends upon the relative severity and depth of the damage. For example, if there is focal damage in the left temporal area, it must be more severe and deeper compared to lesions in other areas to stand out in the localization scale. Perhaps, the Multi-infarct patients of this research do not have severe enough damage in any particular area to be differentiated from the Alzheimer's patients.

The possibility of imprecision in the assessment instrument cannot be ruled out in explaining the absence of localized damage in the MID group. It is conceivable that further medical and psychological research will be able to associate brain lesion sites with specific test behavior through more sensitive instruments. Perhaps identification of lesion sites in different diseases, such as Alzheimer's and Multi-infarct Dementia is possible when specific neuropsychological tests are developed based upon their clinical features. Also, accuracy of medical diagnosis is an important factor in improving the predictive validity of neuropsychological assessment techniques.

However, it must be acknowledged that test behavior is not only a function of brain structures, but is also affected by a variety of personal and social variables (Golden et al., 1982). In sum, the findings of the present research support the argument advanced by other researchers that neuropsychological assessment results must be

evaluated in conjunction with clinical psychosocial and medical data (Golden et al., 1982; Lezak, 1983).

Direction for Future Research

The findings of the present research and the existing literature (see Tierney, Reid, Zorzitto, Snow, Fisher, Campbell-Taylor, & Lewis, 1986) suggest a number of possible research avenues for a better understanding of the brain-behavior relationship in dementia, particularly in Alzheimer's and Multi-infarct disease. First the neuropsychological performance in various stages of the disease should be examined with the help of a comprehensive neuropsychological battery (e.g., LNNB). Thus, it would be interesting to compare the test performance of AD, MID and Depressive patients in their early, middle and late stages. The comparisons may be within or between the groups. Between group comparisons will show whether the three groups differ in the degree and type of deterioration at different stages.

Second, emphasis should be placed on clarifying the diagnostic issues by including even suspected cases of dementia as well as other degenerative diseases (e.g., Multiple Sclerosis, Pick's Disease, Huntington's chorea, etc.) and psychiatric disorders (e.g., Schizophrenia, anxiety disorders, etc.). This will allow better differentiation of cognitive impairment in specific diagnostic groups.

Third, programmatic research is needed to correlate histopathological and neuroradiological data with neuropsychological performance on the one hand, and clinical diagnosis on the other for both dementia and depression groups. Such a multidisciplinary

approach is essential for a differential diagnosis.

Fourth, since most studies in this field are cross-sectional, there seems to be a great need for longitudinal studies. The developmental changes in neuropsychological functioning that occur in Alzheimer's Disease, Multi-infarct Dementia and Depression can be examined adequately through logitudinal studies. The logitudinal studies will also help make better decisions by reducing false positives and false negatives that inevitably occur in the present diagnostic procedure.

Finally, greater attention should be given to both qualitative and quantitative data analyses. For appropriate quantitative analyses, number of subjects in each group is an important consideration. Adequate number of subjects (around 20-30) in each comparison group will allow the use of a variety of multivariate techniques (e.g., Discriminant Functional Analysis). Determination of differential weights to test items for diagnosing different dementia groups has both theoretical and clinical implications. A qualitative analysis of the neuropsychological profile will provide in depth insight into the nature of cognitive impairment.

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APPENDIX A

PERSONAL DATA

Basic Background Information

1. Age: _____ Date of Birth _____

2. Sex: Male _____ Female _____

3. Marital status: Married _____ Single _____ Divorced _____

   Widowed _____ Separated _____ Living Together _____

4. Number of children: _____ son(s) _____ daughter(s) _____

5. Number of grandchildren: _____ grandson(s) _____

   grandaughter(s) _____

6. Birthplace: _____ _____
   city/town country

7. Residence status: citizen _____ permanent resident _____

8. Immigration year: _____ (if applicable)

9. Ethnic background: European _____ Asian _____ Middle Eastern ____

   Native Canadian _____ Others _____

10. Religion _____

11. Education completed:

Elementary	Jr. & High School	College/Univer.	Vocational/ Technical
1 2 3 4 5 6	7 8 9 10 11 12	1 2 3 4 5 6 7 8	1 2 3 4

12. Occupation/Profession (before retirement) _____

13. Occupation/Profession of spouse (before retirement) _____

14. Retirement: Age at retirement _____

    voluntary _____ compulsory

15. Annual Income (including spouse's benefits)

...under $ 5,000 _____ $25,001 - $30,000 _____

$ 5,001 - $10,000 _____ $30,001 - $35,000 _____

$10,001 - $15,000 _____ $35,001 - $40,000 _____

$15,001 - $20,000 _____ $40,001 - $45,000 _____

$20,001 - $25,000 _____ $45,001 & over... _____

16. Residence:

House _____ Condominium _____ Apartment _____

owned _____ rented _____

Nursing Home _____ Home for aged _____ Hospital _____

Extended Care Centre _____ Others _____

17. Transportation and mobility:

Car _____ Public transportation _____ Others _____

18. Interests and Hobbies:

sports _____

commercial recreation (movies, theatre, etc.) _____

gardening _____ crocheting _____ knitting _____

reading _____ travelling _____ listening to music _____

T.V. watching _____ pet(s) _____ Others _____

19. Community involvement:

volunteer work _____

community centers _____

Senior citizen centers _____

church group _____

others _____

20. Contact with family members:

At least once a week _____ At least once every two weeks _____

At least once a month _____ At least once every 3 months _____

At least once every 6 months _____ At least once a year _____

21. Most frequent social interactions with:

sons _____ daughters _____ grandsons _____ granddaughters ____

brothers _____ sisters _____ other relatives _____

friends _____

22. Medical history:

Date of your last medical checkup _____

How do you rate your general medical health?

Excellent _____ Good _____ Fair _____ Poor _____

Have you ever had or do you have any of the following?

Heart disease: yes _____ no _____

If yes, please specify _____

High blood pressure: yes _____ no _____

If yes, please specify _____

Stroke: yes _____ no _____

If yes, please specify _____

Diabetes: yes _____ no _____

If yes, please specify _____

Hypertension: yes _____ no _____

If yes, please specify _____

Epilepsy or Seizures: yes _____ no _____

If yes, please specify _____

Fainting spells: yes _____ no _____

If yes, please specify _____

Parkinsonism: yes _____ no _____

If yes, please specify _____

Huntington's chorea: yes _____ no _____

If yes, please specify _____

Alzheimer's disease: yes _____ no _____

If yes, please specify _____

Thyroid problems: yes _____ no _____

If yes, please specify _____

Others: yes _____ no _____

If yes, please specify _____

23. Psychiatric information:

Have you ever seen a spychologist or a psychiatrist?

yes _____ no _____

If yes, please specify _____

24. Medication Record:

Are you on any kind of prescribed medication at present?

yes _____ no _____

If yes, please specify _____

APPENDIX B

Dear Dr.

I am a doctoral student in the Department of Educational Psychology at the University of Alberta. My dissertation research is concerned with the neuropsychological functioning of the elderly. I am conducting this research under the supervision of a number of faculty members at the University of Alberta.

The main purpose of my study is to examine the nature of neuropsychological changes in the elderly suffering from Alzheimer's disease, Multi-infarct dementia and depression. I will be using a number of psychological tests which would take approximately 4-5 hours for each participant to complete. The testing will be done in several short sessions depending upon the mental and physical conditions of the participant. The psychological tests to be used in this study contain items such as answering simple questions, simple writing, and performing simple manual tasks. The test items do not require any physical strength on the part of the participants, and are not harmful--physically or mentally. These tests will not interfere with the treatment and management plans of the patient.

All tests will be administered by this researcher or trained research assistants. Thus, this research will not involve any staff or equipment of the hospital, except occasional supervision of the attending physicians or other supporting staff members. Please note that the test results will be kept completely confidential and will be used only for research purposes by this investigator.

I would like to take this opportunity to draw your attention to the fact that the proposed research is likely to contribute a great deal to our understanding of the neuropsychological changes in old persons suffering from selected degenerative disease process. In addition, this research will be helpful in diagnosis, treatment, management and rehabilitation of those elderly who require such services.

I am, therefore, requesting your help and cooperation in providing suitable elderly patients for psychological assessment. If you have any questions, please feel free to call me at 435-5160 or 432-3226.

Thank you very much for your time and interest in this project.

Yours sincerely,

Asha Sinha

APPENDIX C

Dear Sir/Madam

I am a doctoral student in the Department of Educational Psychology at the University of Alberta. For my Ph.D. degree, I am doing research on the psychological functioning of the elderly persons under the supervision of staff physicians and university professors.

The main purpose of my research is to study the nature of psychological changes in those elderly who are suffering from Alzheimer's disease, Multi-infarct dementia and depression. I will be using a number of psychological tests which will take approximate 4-5 hours to complete. In order to avoid any physical or mental strain in the patients, the testing will be conducted in several small sessions. The testing consists of answering simple questions, simple writing, and performing simple manual tasks. These psychological tests are completely harmless. The welfare of the patients is further protected by the fact that they are always under the supervision of medical doctors.

The test results will be kept completely confidential, and will be available only to the attending physicians. The findings of this research will be useful to the doctors in making diagnosis, and providing adequate treatment, management and rehabilitation programmes. I am, therefore, requesting your help in completing this research project. I would appreciate very much if you would kindly sign the attached consent form. If you have any questions, please feel free to contact me at 435-5160 or 432-3226.

Thank you very much for your cooperation.

Sincerely,

Asha Sinha

CONSENT FOR

PSYCHOLOGICAL TESTING

I, _____ have no objection in taking the
patient's name

required psychological tests for the research project of Asha Sinha, a doctoral student at the University of Alberta. I have read the researcher's letter of intent explaining the nature and purpose of the research project. I understand that the test data will be kept confidential and will not be released to any one other than the researcher and the attending physician without my permission. I also understand that I may withdraw from the testing at any time.

Signed: _____
(patient)

Witness: _____

Date: _____

CONSENT FOR

PSYCHOLOGICAL TESTING

I, _____ have no objection to
(guardian's name)

_____ taking the required psychological
(patient's name)

tests for the research project of Asha Sinha, a doctoral student at the University of Alberta. I have read the researcher's letter of intent explaining the nature and purpose of the research project. I understand that the test data will be kept confidential and will not be released to any one other than the researcher and the attending physician without my permission. I also understand that my ward _____ may withdraw from the testing at any time.
(patient's name)

Signed: _____
(guardian)

Witness: _____

Date: _____